This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

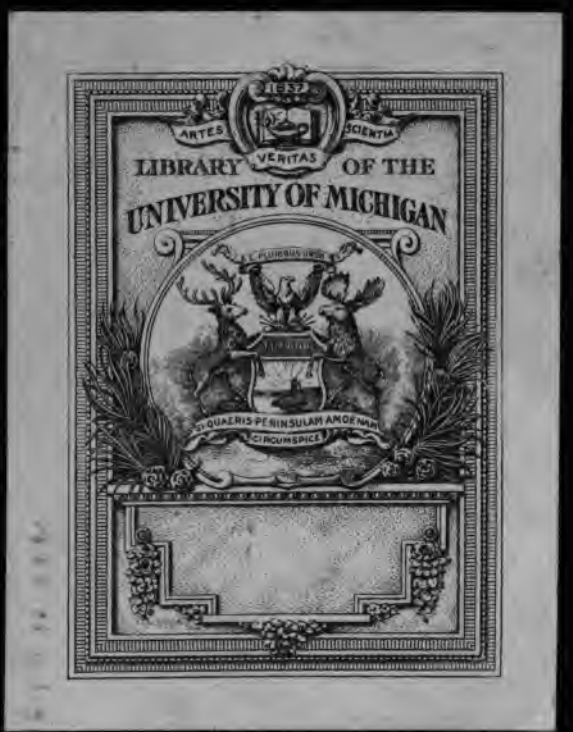
+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

A 3 9015 00380 260 3
University of Michigan - BUHR

TRANSACTIONS
OF THE
EPIDEMIOLOGICAL SOCIETY
OF
LONDON


ARTES
VERITAS
SCIENTIA
LIBRARY OF THE
UNIVERSITY OF MICHIGAN
E PLURIBUS UNUM
TUEBOR
SI QUAERIS PENINSULAM AMOENAM
CIRCUMSPICE

610.3
E64

TRANSACTIONS

OF THE

EPIDEMIOLOGICAL SOCIETY

OF

LONDON.

NEW SERIES. VOL. XXIV.

SESSION 1904-1905.

LONDON:
WILLIAMS AND NORGATE,
14, HENRIETTA STREET, COVENT GARDEN, LONDON, W.C.

MDCCCCV.

All rights reserved.

LONDON:
PRINTED AT THE BEDFORD PRESS, 20 AND 21, BEDFORDBURY, W.C.

CONTENTS.

PAGE

The Epidemiological Aspects of Industrial Diseases. By B. A. WHITELEGGE, C.B., M.D., F.R.C.P. 1

Ticks and Tick-Transmitted Diseases. By GEORGE H. F. NUTTALL, M.A., M.D., Ph.D., F.R.S. 12

Discussion on Dr. Nuttall's Paper, by Sir PATRICK MANSON, Dr. THEODORE THOMSON, Dr. BENTLEY, Professor W. J. SIMPSON, Dr. G. C. LOW. Dr. NUTTALL's reply 26-32

A Contribution to the Etiology of Rheumatic Fever. By G. B. LONGSTAFF, M.D., F.R.C.P. 33

Discussion on Dr. Longstaff's Paper, by The PRESIDENT, Dr. BULSTRODE, Sir WILLIAM CHURCH, Dr. NEWSHOLME, Dr. HAMER, Dr. BOND, Dr. J. T. C. NASH. Dr. LONGSTAFF's reply 63-83

On the Relation of the Parasitic Protozoa to each other and to Human Disease. By E. J. MCWEENEY, M.A., M.D., D.P.H., F.R.C.P.I. ... 84

Discussion on Dr. E. J. McWeeney's Paper, by Sir PATRICK MANSON, Dr. COPEMAN, The PRESIDENT. Prof. MCWEENEY's reply 105-112

Ankylostomiasis. By A. E. BOYCOTT, M.A., M.D., B.Sc. 113

Discussion on Dr. Boycott's Paper, by Sir PATRICK MANSON, Dr. LEIPER, Prof. KENWOOD, Dr. LOW. Dr. BOYCOTT's reply 130-142

On the Spread of Small-Pox Occasioned by Small-Pox Hospitals during the Epidemic Period, 1900-1904; and its Relation to Atmospheric Convection. By G. S. BUCHANAN, M.D. 143

Discussion on Dr. Buchanan's Paper, by Dr. C. B. KER, Dr. NEWSHOLME, Dr. NIVEN, Dr. HOPE, Dr. MCVAIL, Dr. R. J. REECE, Dr. PHILIP BOOBBYER, Dr. J. THRESH, Dr. G. REID, Dr. R. D. SWEETING, Prof. KENWOOD, Dr. WELLESLEY HARRIS, Lieut.-Col. A. M. DAVIES, Dr. J. T. C. NASH, Dr. HAMER, Dr. BULSTRODE, Dr. CHALMERS. Dr. BUCHANAN's reply 174-258

Phthisis Rates: Their Significance and their Teaching. By ARTHUR RANSOME, M.D., F.R.S. 259

Discussion on Dr. Ransome's Paper, by The PRESIDENT, Sir SHIRLEY MURPHY, Dr. NEWSHOLME, Dr. SEATON, Dr. ALLAN, Dr. BULSTRODE. Dr. RANSOME's reply ... 285-294

Ancient Theories of Causation of Fever by Mosquitoes 295

148373

EPIDEMIOLOGICAL SOCIETY OF LONDON.

OFFICERS AND OTHER MEMBERS OF COUNCIL

FOR THE SESSION 1904-1905.

President.

B. A. WHITELEGGE, C.B., M.D., F.R.C.P.

Honorary Vice-Presidents.

THE RIGHT HON. THE EARL OF ABERDEEN, Haddo House, Tarland, Aberdeenshire.

THE RIGHT HON. THE EARL OF MEATH, 83, Lancaster Gate.

THE RIGHT HON. THE LORD LISTER, F.R.S., 12, Park Crescent, Portland Place, W.

Vice-Presidents.

Sir JOSEPH FAYRER, Bart., K.C.S.I., LL.D., M.D., F.R.S., Q.H.P., 15, Devonshire Street, Portland Place.

Sir J. BURDON SANDERSON, Bart., M.D., F.R.S., 64, Banbury Road, Oxford.

Sir JOSEPH EWART, M.D., F.R.C.P., Dep. Surgeon-General, Indian Medical Department (*retired*), Bewcastle, Dyke Road, Brighton.

J. F. PAYNE, M.A., M.D., F.R.C.P., 78, Wimpole Street, W.

Sir SHIRLEY F. MURPHY, 9, Bentinck Terrace, Regent's Park, N.W.

JOHN TATHAM, M.D., F.R.C.P., Somerset House, W.C.

J. LANE NOTTER, M.A., M.D , Colonel R.A.M.C., 3, Kensington Mansions, Earl's Court, S.W.

H. FRANKLIN PARSONS, M.D., Local Government Board, S.W.

Sir PATRICK MANSON, K.C.M.G., F.R.S., M.D., F.R.C.P., LL.D., 21, Queen Anne Street, Cavendish Square, W.

Treasurer.

R. D. R. SWEETING, M.A., M.D., 34, Carlisle Mansions, Westminster, S.W.

General Secretaries.

H. TIMBRELL BULSTRODE, M.A., M.D., 4, The Mansions, Earl's Court, S.W.

W. H. HAMER, M.A., M.D., F.R.C.P., 55, Dartmouth Park Hill, N.W.

Secretary for Navy.

Deputy Inspector-General THEODORE PRESTON, R.N., 16, St. John Park, Blackheath, S.E.

Secretary for Army.

Vacant.

Foreign and Colonial Secretaries.

Italy	E. F. Willoughby, M.D. Angelo Celli, M.D.
China	J. A. Lowson.
Cape Colony	A. J. Gregory, M.D.
Transvaal	G. Turner, M.D. W. C. Pakes, M.D.
West Africa	W. T. Prout, M.D.
Congo Land	A. Sims, M.D.
British East Africa	R. W. Moffat, C.M.G., M.D.
Natal	O. J. Currie, M.D.
Orange River Colony ...	Dr. Yule.
France	B. A. Whitelegge, C.B., M.D., F.R.C.P.
Germany	S. Monckton Copeman, M.D., F.R.C.P., F.R.S.
Japan	Professor S. Kitasato.
Sweden	Klas Linroth, M.D.
Norway	Axel Holst, M.D.
New South Wales	F. Tidswell, M.D.
Portugal	A. H. Shadwell, M.B.
West Indies	G. C. Henderson, M.D.
United States of America—North	Sir Joseph Ewart, M.D., F.R.C.P. Surgeon-General J. Billings, M.D.
Bengal	J. Neild Cook, M.D.
Bombay	Major Leslie, R.A.M.C.
Madras	Colonel King, M.D.
Ceylon	Sir Allan Perry, M.D.
Straits Settlements	E. A. O. Travers.
Cuba	Dr. Guiteras.
Canada	Dr. Montizambert. Dr. Adami.
Polynesia	Bolton G. Corney.
Russia	Frank G. Clemow, M.D.
Holland and Belgium ...	V. R. N. Saltet, M.D.
Spain	Phillip Hauser, M.D.
Egypt and the Red Sea ...	Armand Ruffer, M.D. Sir H. Pinching, K.C.M.G.
United States of America—South	R. Bruce Low, M.D. T. R. Davison, M.D.
Brazil	Professor Magalhaes.
Argentine Republic	Samuel Gache, M.D.

Other Members of Council.

Sidney Coupland, M.D., F.R.C.P. (S. 1894-98.)
E. W. Goodall, M.D.
E. O. Hopwood, M.A., M.D. (S. 1890-94.)
J. C. McVail, M.D.
Sidney Martin, F.R.S., M.D., F.R.C.P.
A. Newsholme, M.D., F.R.C.P.
Charles E. Paget. (S. 1883-90.)
Louis Parkes, M.D. (S. 1889-91.)
Staff-Surgeon W. E. Home, M.D., R.N.
Meredith Richards, M.D.
K. W. Goadby.

LIST OF MEMBERS.*

(P) *President*; (T) *Treasurer*; (S) *Secretary*.

Honorary Members.

Elected
1888. Abbate Pacha, Cairo.
1888. Amada, J. J. da Silva, M.D., Lisbon.
1888. Bacelli, Guido, M.D., Rome.
1888. Billings, Dr. J. S., Director New York Public Library, 40, Lafayette Place, New York City, U.S.A.
1888. Brouardel, S., M.D., 195, Boulevard St. Germain, Paris.
1900. Fayrer, Sir Joseph, Bart., K.C.S.I., LL.D., M.D., F.R.S., Q.H.P., 15, Devonshire Street, Portland Place, W.
1868. Guipon, D. J., M.D., Laon, Aisne, France.
1885. Humphreys, Noel A., General Register Office, Somerset House, W.C.
1888. Koch, R., M.D., 36, Klosterstrasse, Berlin.
1888. Larrey, F. H., M.D., 91, Rue de Lille, Paris.
1888. MacLaurin, H. A., M.D., Macquarie Street, Sydney, N.S.W.
1896. Monod, H., Ministère de l'Interieur, Paris.
1894. Müller, Coleman, M.D., St. Rochus Hospital, Buda-Pesth.
1904. Pistor, Moritz, M.D., 15 Pariser Strasse 3, Berlin W.
1900. Salmon, E., M.D., United States Department of Agriculture, Bureau of Animal Industry, Washington, U.S.A.
1883. Sonsino, Prospero, M.D., 11, Via San Lorenzo, Pisa.
1888. Sternberg, Geo. M., M.D., Johns Hopkins University, Baltimore, U.S.A.
1888. Vallin, E., M.D., 5, Avenue de Messine, Paris.

Ordinary Members.

1892. Aldwinkle, T. W., F.R.I.B.A., F.S.S., 20, Denman Street, S.E.
1900. Argles, Frank, Hermon Lodge, Wanstead, N.E.
1882. Armstrong, Henry E., D. Hy., Town Hall, Newcastle-on-Tyne.
1888. Ashby, Alfred, M.B., Town Hall, Reading, Berks.
1884. Atkinson, F. E., "Bowerley," Settle, Yorkshire.
1901. Balfour, Andrew, M.D., B.Sc., Gordon College, Khartoum.
1896. Beggs, J. E., M.D., Grove Hospital, Tooting Grove, Lower Tooting, S.W.
1903. Beveridge, W. W. O., Major, D.S.O., R.A.M.C., 25, Charles Street, St. James' Square, S.W.
1897. Bond, F. T., M.D., 3, Beaufort Buildings, Gloucester.
1892. Bond, W. A., M.D., Public Health Department, Holborn.
1889. Boobbyer, Philip, M.B., Municipal Offices, Nottingham.
1904. Branthwaite, R. W., M.D., D.P.H., Home Office, Whitehall, S.W.
1903. Brincker, J. A. H., M.B., Borough Fever Hospital, Croydon.
1889. Brock, J. H. E., M.D., 77, Fellows Road, N.W.
1903. Brodie, Sir Benjamin V. S., Bart., M.A., J.P., Brockham Warren, Betchworth, Surrey.

---

* Members are requested to inform the Secretaries of any corrections when necessary.

Elected

1905. Brown, W. Carnegie, M.D., 13, Hampstead Hill Gardens. N.W.
1891. Bruce, R. M., Western Hospital, Fulham.
1894. Buchanan, G. S., M.D., Local Government Board, S.W.
1889. Bulstrode, H. Timbrell, M.D. (*Hon. Secretary*), Local Government Board, S.W.
1903. Butler W., M.B., Municipal Offices, Willesden.
1888. Caiger, F. Foord, M.D., F.R.C.P., South Western Hospital, Stockwell. [S.W.
1904. Cameron, A. F., M.D., South Western Hospital, Stockwell,
1900. Cameron, Allan, M.D., 3, Brick Court, Temple, E.C.
1897. Cantlie, James, M.B., F.R.C.S., 46, Devonshire Street, Portland Place, W.
1900. Chalmers, A. K., M.D., D.P.H., 7, Buckingham Terrace, Hillhead, Glasgow.
1897. Childs, Christopher, M.D., 10, Manchester Square.
1892. Clemow, Frank Gerard, M.D., British Embassy, Constantinople.
1897. Collingridge, W., M.D., Guildhall, E.C.
1903. Colwell, Hector Alfred, M.B., D.P.H., 55, Ridge Road, Hornsey.
1891. Copeman, S. M., M.D., F.R.C.P., F.R.S., Local Government Board, S.W.
1904. Coplans, Myer, M.D., Stroud, Gloucestershire.
1892. Coupland, Sidney, M.D., F.R.C.P., 16, Queen Anne Street, W. (S. 1894-98).
1900. Cowburn, D. A., M.D., 31, Barkston Gardens, S.W.
1902. Crawford, G. S., D.P.H., Major R.A.M.C., 21, Grosvenor Road, Aldershot.
1892. Crosby, H., M.B., 19, Gordon Square, W.C.
1903. Daniels, C. W., M.B., Medical Superintendent, London School of Tropical Medicine, Albert Docks, E.
1900. Davies, A. M., Lt.-Col. R.A.M.C., 64, West Park, Eltham, Kent.
1892. Dickson, T. Hugh, M.B., Custom House, Lower Thames Street., E.C.
1876. Downes, A. H. J., M.D., Local Government Board, S.W.
1895. Dowson, W., M.D., Newcome, Stanley Road, Sutton.
1895. Dudfield, R., M.B., 19, Blomfield Road, Maida Vale, W.
1898. Eaton, Oliver, Wescott, Exmouth.
1879. Ewart, Sir Joseph, M.D., Dep. Surg.-Gen. Ind. Med. Dept. (*retired*), Bewcastle, Dyke Road, Brighton. (P. 1890-92.)
1903. Farrar, R. A., M.A., M.D., Local Government Board, S.W.
1900. Firth, R. H., F.R.C.S., D.P.H., Professor of Military Hygiene, Army Hygiene Laboratory, Examination Hall, Embankment, W.C.
1892. Fletcher, W. W. E., M.B., Local Government Board, S.W.
1903. Freeman, E. C., M.R.C.S., L.R.C.P., Major R.A.M.C., care of Holt and Co., 3, Whitehall Place, S.W.
1903. Fremantle, F. E., M.A., M.B., The College, Guy's Hospital, S.E.
1899. Fremlin, H. S., Government Lymph Laboratories, Chelsea Bridge, S.W.
1894. Fuller, Andrew, Local Government Board, S.W.
1900. Goadby, Kenneth Weldon, 21, New Cavendish Street, W.
1891. Goodall, E. W., M.D., Eastern Hospital, Homerton.
1904. Grünbaum, A. S. F., M.D., F.R.C.P., 67, Delia, The Drive, Roundhay, Leeds.
1892. Hamer, W. H., M.D., F.R.C.P., 55, Dartmouth Park Hill, N.W. (*Hon. Secretary.*)

Elected

1898. Hardy, F. W., M.B., B.C., D.P.H., Major R.A.M.C., 3, Whitehall Place, S.W.
1899. Harford, C. F., M.D., Livingstone College, Leyton, E.
1894. Harris, W., M.D., Town Hall, Catford, S.E.
1898. Harwood, J. G., Surg.-Major, F.R.C.S., A.M.S., 17, Whitehall Place, S.W.
1887. Home, W. E., Fleet Surg., R.N., M.D., B.Sc., H.M.S. Exmouth.
1900. Hope, Edward Wm., M.D., D.Sc., Municipal Offices, Liverpool.
1883. Hopwood, E. O., M.D. (S. 1890-94), London Fever Hospital, Islington, N.
1903. Houston, A. C., M.B., C.M., D.Sc., 14, Upper Addison Gardens, Kensington, W.
1892. Hume, F. N., Northern Hospital, Winchmore Hill.
1891. Hunter, W., M.D., 54, Harley Street, W.
1897. Hunting, W., F.R.C.V.S., 16, Trafalgar Square, Chelsea, S.W.
1887. James, C. A., D.P.H., 49, Stamford Hill, N.
1899. Johnstone, Ralph, M.D., Local Government Board, S.W.
1896. Jones, Herbert, Bankside, Hereford.
1900. Kaye, J. R., M.B., Wentworth Lodge, Wakefield.
1892. Kenwood, H. R., M.B., 9, Alexander Villas, Finsbury Park, N.
1900. Ker, C. B., M.D., City Hospital, Edinburgh.
1895. Klein, E., M.D., F.R.S., Harewood, Riverdale Gardens, Twickenham Park, Richmond.
1895. Legge, T. M., M.D., Home Office, S.W.
1895. Littlejohn, H., M.D., 1, Atholl Terrace, Edinburgh.
1903. Logan, T. Stratford, West Riding Asylum, Wakefield.
1880. Longstaff, G. B., M.D., Highlands, Putney Heath, S.W.
1901. Lovell, Sir Francis, C.M.G., London School of Tropical Medicine, Albert Docks, E.
1903. Low, George Carmichael, M.B., London School of Tropical Medicine, Albert Docks, E.
1887. Low, R. Bruce, M.D., Local Government Board, S.W.
1904. McCleary, G. F., M.D., D.P.H., Town Hall, Battersea.
1878. MacCombie, John, M.D., Brook Hospital, Shooter's Hill.
1903. McCrindle, J. D., M.B., Council House, Birmingham.
1903. McCulloch, T., M.B., C.M., Dep. Assistant Director.-Gen., 56, West Park, Eltham.
1094. McFadden, A. W. J., M.B., D.P.H., 326, Lower Fore Street, Edmonton, Middlesex.
1905. Macfadyen, Professor Allan, M.D., 14, Daleham Gardens, Hampstead.
1904. McManus, L. S., Mayo House, Spencer Park, Wandsworth Common, S.W.
1889. McVail, J. C., M.D., 24, George Square, Glasgow.
1894. Mair, L. Darra, M.D., Local Government Board, S.W.
1894. Manby, P., M.D., Local Government Board, S.W.
1900. Manson, Sir Patrick, K.C.M.G., M.D., F.R.S., 21, Queen Anne Street, Cavendish Square, W. [P. 1900-1902.]
1885. Martin, Sidney, F.R.S., M.D., F.R.C.P., 10, Mansfield Street, W.
1896. Mivart, F. St. George, M.D., Local Government Board, S.W.
1875. Murphy, Sir Shirley Forster, 9, Bentinck Terrace, Regent's Park, N.W. (S. 1877-89. P. 1894-96.)
1903. Nash, J. T. C., M.D., D.P.H., 4, Argyle Road, Southend.
1896. Newman, G., M.D., 2, Woburn Square, W.C.
1891. Newsholme, A., M.D., F.R.C.P., Town Hall, Brighton.

Elected

1897. Niven, James, M.B., Town Hall, Manchester.
1889. Notter, J. Lane, M.D., Colonel, R.A.M.C., 3, Kensington Mansions, Earl's Court, S.W. (P. 1896-98.)
1903. Nuttall, G. H. F., M.A., M.D., Ph.D., F.R.S., 3, Cranmer Road, Cambridge.
1899. O'Connor, J. E., Captain, R.A.M.C., White Lodge, Kirby Muxloe, Leicester.
1883. Paget, Charles E., County Hall, Northampton. (S. 1883-90.)
1884. Parkes, Louis, M.D., 61, Cadogan Square, S.W. (S. 1889-91.)
1883. Parsons, H. Franklin, M.D., Local Government Board, S.W. (P. 1898-1900.)
1880. Payne, J. F., M.D., F.R.C.P., 78, Wimpole Street, W. (P. 1892-94).
1889. Preston, Theodore J., Deputy Inspector-General, R.N., 16, St. John's Park, Blackheath, S.E.
1892. Reece, R. J., M.D., Local Government Board, S.W.
1894. Reid, G., M.D., Eastgate House, Stafford.
1896. Richards Meredith, M.D., B.S., Town Hall, Croydon.
1875. Roberts, F. T., M.D., F.R.C.P., 102, Harley Street, W.
1898. Robertson, J., M.D., Town Hall, Birmingham.
1899. Ruffer, Marc Armand, M.D., Ramleh, Egypt, President of the Sanitary Maritime and Quarantine Council, Alexandria, Egypt.
1895. Saltet, R. H., M.D., University of Amsterdam.
1873. Seaton, E. C., M.D., F.R.C.P., 93, St. George's Square, S.W.
1899. Shadwell, A. H., M.B., Burnham, Bucks.
1903. Skinner, B.M., Lieut.-Col. R.A.M.C., 68, Victoria Street, S.W.
1899. Snell, E. H., M.D., 80, Hay Lane, Coventry.
1899. Steegman, E., M.B., Eamont Lodge, Spring Grove, Isleworth.
1896. Stewart, C. H., Professor, M.B., D.Sc., F.R.S., Edin., Public Health Laboratory, Edinburgh University.
1892. Stewart, W. J. J., M.D., The Hospital, Neasden, W.
1898. Stott, Hugh, The Hermitage, Lewes, Sussex.
1880. Sweeting, R. D. R., M.A., M.D., Local Government Board, S.W. (*Treasurer*).
1888. Sykes, J. F. J., D.Sc., M.D., 40, Camden Square, N.W.
1880. Tatham, J., M.D., F.R.C.P., Somerset House, W.C.
1905. Thomas, C. J., M.B., B.Sc., 37, Mount Nod Road, Streatham.
1888. Thomson, Theodore, M.D., Local Government Board, S.W.
1892. Thomson, Fred. Holland, M.B., Gore Farm Hospital, near Dartford.
1889. Thresh, John C., M.B., D.Sc., Town Hall, Chelmsford.
1901. Tidswell, F., M.B., D.P.H., care of The Agent-General for New South Wales, Victoria Street.
1903. Todd, Chas., M.D., D.P.H., Sanitary Department, Cairo, Egypt.
1896. Turner, F. Meadows, M.D., South Eastern Hospital, Deptford.
1903. Wanklyn, W. McC., D.P.H., 8, St. Martin's Place, W.C.
1892. Wheaton, S. W., M.D., Local Government Board, S.W.
1882. Whitelegge, C.B., M.D., F.R.C.P., Home Office, S.W. (*President.*)
1897. Williams, Herbert, M.D., 51, King William Street, Greenwich, London, S.E.
1894. Williams, W., M.B., County Offices, Cardiff.

Elected

1883. Willoughby, Edward F., M.D., Bratton Lodge, Green Lanes, N.
1905. Wright, Oswald Kentish, B.A., 113, Elm Park Mansions, S.W.
1883. Young, Keith Downes, F.R.I.B.A., 17, Southampton Street, W.C.
1895. Young, C. W. F., M.D., Guildhall, Westminster, S.W.

Non-Resident Subscribing Members.

(Law III, 8.)

1875. Armistead, W., M.B., Stapleford, Cambridge.
1894. Cameron, Spottiswoode, M.D., Municipal Buildings, Leeds.
1902. Clayton, F. H. A., M.D., H.M.S. "Prometheus," c/o The Admiralty, S.W.
1889. Cooper, C. H., A.M.I.C.E., Local Board Offices, Wimbledon.
1891. Davies, D. S., M.D., Sanitary Offices, Bristol.
1895. Dixey, H. E., M.D., Woodgate, Great Malvern.
1902. Elkington, J. S. C., M.D., Office of Chief Medical Officer, Hobart, Tasmania.
1894. Evans, W. Arnold, M.D., Town Hall, Bradford.
1897. Fraser, A. M., M.D., Town Hall, Portsmouth.
1903. Hendley, A. G., Major, I.M.S., c/o Grindlay & Co., Bombay.
1892. Horne, T., M.D., Town Hall, Stockton on-Tees.
1889. James, A. W., 69, Gloucester Terrace, Hyde Park, W.
1902. Lewis, C. J., M.D., D.Sc. 13, Walker Street, Edinburgh.
1899. Low, J. Spencer, M.B., Helmsley House, Christchurch Road, Tulse Hill.
1897. Lowson, J. A., care of Mrs. Grant, Craiglachie, Keswick Road, Putney, S.W.
1895. Mitchell, Trafford, M.D., Gorseinon, Glamorganshire.
1872. Moore, Sir J. W., M.D., 40, Fitzwilliam Square West, Dublin.
1899. Moores, S. G., Surgeon-Major, Connaught Hospital, South Farnborough.
1894. Pattin, H. Cooper, M.D., Municipal Offices, Norwich.
1875. Ransome, Arthur, M.D., F.R.S., Sunnyhurst, Dean Park, Bournemouth.
1898. Rogers, Leonard, M.D., F.R.C.S., I.M.S. Capt.; care of Thos. Cook and Son, 9, Old Court House, Calcutta.
1888. Walford, Edward, M.D., Municipal Offices, Cardiff.
1894. Weston, G. H., M.D., Shirley, Southampton.
1898. Williams, H. J. E. H., M.D., City Hospital, Winter Street, Sheffield.
1888. Wilson, J. Mitchell, M.D., County Hall, Beverley, Yorks.

Non-Resident Members.

1896 Adams, P. T., M.D., Bloemfontein, O.R.C.
1903. Armstrong, W. G., M.B., Department of Public Health, City Town Hall, Sydney, N.S.W.
1894. Atkinson, M., M.D., Hong Kong.
1888. Bately, J., M.D., High Street, Gorleston, Great Yarmouth.
1895. Borthwick, T., M.D., Kensington, Adelaide, South Australia.
1883. Bovill, Surg. Lieut.-Col. Edward, M.D., C.M., I.M.S., Bengal, India (*retired*).
1889. Bruce, David, M.B., Lieut.-Col. R.A.M.C., c/o War Office.

Elected

1864. Buckle, Fleetwood, M.D., 20, Merton Road, Southsea (*retired*).
1904. Bushnell, F. G., M.D., D.P.H., Sussex County Hospital, Brighton.
1866. Caddy, J. T., M.D., Dep. Inspec.-Gen., The Charterhouse, Lion Gate Gardens, Richmond, Surrey.
1887. Collins, G. W., The Mall, Wanstead, N.E.
1885. Corney, Bolton G., Suva, Fiji; and 26, Bedford Place, Russell Square, W.C.
1885. Crawford, D. G., Surg.-Major I.M.S., Mymensingh, Lower Bengal.
1891. Crook, H. E., M.D., 23, Dalby Square, Cliftonville, Margate.
1876. Day, E. J., Dorchester.
1886. Donovan, Justin F., M.D., Port Royal, Jamaica.
1898. Eaton, Oliver, Westcott, Exmouth.
1884. Faulkner, A. S., Surg. Major Bombay Army, Messrs. Grindlay and Co., 55, Parliament Street, S.W.
1888. Fosbroke, G. H., M.D., Shire Hall, Worcester.
1890. Garrett, J. H., M.D., Municipal Offices, Cheltenham.
1876. Hardwicke, H. Junius, M.D., 13, Magdalen Terrace, St. Leonard's-on-Sea.
1880. Harrison, R. Charlton, M.D., 19, Uxbridge Road, Ealing, W.
1881. Henderson, G. C., M.D., Kingston, Jamaica.
1882. Herring, John F., St. Mary's House, Atherstone, Warwickshire.
1875. Hime, T. W., M.D., 54, Horton Road, Bradford.
1875. Home, Sir Anthony D., K.C.B., V.C., M.D., Surg.-Gen. (*retired*) 9, Denmark Terrace, Brighton.
1881. Irwin, Chauncey Graves, M.D. (*retired*), Dep.-Surg.-Gen., Casa Hatfield, St. Albans, Herts.
1887. Kelly, C., M.D., Gratwicke Road, Worthing.
1886. Lingard, A., M.B., Imperial Bacteriologist, Muktesar, Kumâon Hills, N.W.P., India.
1881. Lyons, R. T., M.D., Brig.-Surg. (*retired*), 9, Scroope Terrace, Trumpington Street, Cambridge.
1860. May, George, M.B., F.R.C.S. (*retired*), The Warren, Caversham, Reading
1899. Millard, C. K., M.D., B.Sc., Town Hall, Leicester.
1890. M'Neill, R., M.D., Argyll County Council, Oban, N.B.
1900. Moffet, G. E., Surg.-Major, R.A.M C., 5, Castle Street, Brechin, Forfarshire.
1881. Oldham, Chas., Brigade-Surg. Ind. Med. Dept. (*retired*), The Lodge, Great Bealings, Woodbridge, Suffolk.
1897. Pakes, W. C , M.D., Government Laboratory, Pretoria, South Africa.
1874. Reid, Walter, M.D., Dep. Inspector-General, Royal Naval Hospital, Haslar (*retired*).
1863. Rowe, T. Smith, M.D., 1, Union Crescent, Margate.
1881. Russell, E. G., M.B., B.Sc., Surg. Lieut.-Col., I.M.S. Bengal Army (*retired*).
1859. Sanderson, Sir J. Burdon, Bart., M.D., F.R.S., 64, Banbury Road, Oxford.
1884. Simpson, W.J., M.D., King's College.
1903. Stokes, E. S., M.B., Department of Public Health, Sydney, N.S.W.
1889. Street, A. F., M.D., St. Mildred's Road, Westgate-on-Sea, Kent.
1885. Sturridge, P. F., Stramongate, Kendal, Westmorland.

Elected

1885. Tomes, Arthur, Surg. Lieut.-Col. Ind. Med. Service, St. Leonard's, Malvern.
1876. Turner, G., M.D., Cape Town, South Africa.
1893. Whiston, P. H., Surgeon-Major, R.A.M.C., Junior United Service Club, S.W.; Charles Street, St. James', S.W.
1884. Williamson, R. E., M.B., Romagna House, Otley, Yorks.

Corresponding Members.

1894. Catrin, L., Dr., Val-de-Grâce, Paris.
1856. Cox, Wm. Isidore, Hawkesbury-Upton, Chippenham, Gloucestershire.
1871. Cuningham, J. M., M.D., C.S.I., Surg.-Gen. Ind. Med. Dept., Sanitary Commissioner of Govt. of India (*retired*).
1894. Davidson, Dr., 30, Morningside Place, Edinburgh.
1878. Delmas, Louis H., M.D., P.O. Box 524, Havana, Cuba.
1895. Gache, Samuel, M.D., Buenos Ayres, Argentine Republic.
1892. Hauser, Philip, M.D., Madrid.
1889. Hewitt, Chas. N., M.D., Red Wing, Minnesota, U.S.A.
1900. Holst, Axel, M.D., Professor of Hygiene in the University of Christiania, Norway.
1857. Leaõ, Jose Fernandes da Silva, Cape de Verde.
1900. Linroth, Klas, M.D., Medical Officer of Health of Sweden, Stockholm.
1900. Mosny, Ernest, M.D., Médecin des Hôpitaux de Paris, Comité Consultatif d'Hygiene Publique de France.
1894. Netter, Dr. Arnould, 129, Boulevard St. Germain, Paris.
1854. Purple, S. S., M.D., New York, U.S.A.
1900. Reincke, J. T., M.D., Chief M.O.H. of Hamburg, Holzdamm 26, Hamburg.
1886. Rochard, G., M.D., Paris.
1900. Schierbeck, Niels Peter, M.D., Chief of the Hygiene Laboratory, University of Copenhagen, Denmark.
1873. Schleisner, —, M.D., Copenhagen.
1874. Schlimmel, —, M.D., Teheran.
1888. Thompson, Ashburton, M.D., 93, Macquarie Street, Sydney, New South Wales.
1861. Whitelaw, Wm., M.D., Kirkintilloch, Dumbartonshire.

Jenner Medallists.

1898. William Henry Power, C.B., F.R.S., Chief Medical Officer of the Local Government Board, Whitehall, S.W.
1902. A. Laveran, Membre de l'Institut et de l'Academie de Medicine, 25, Rue du Montparnasse, Paris.

TRANSACTIONS

OF

The Epidemiological Society of London.

INAUGURAL ADDRESS OF SESSION 1904-1905.

THE EPIDEMIOLOGICAL ASPECTS OF INDUSTRIAL DISEASES.

BY B. A. WHITELEGGE, C.B., M.D., F.R.C.P.,
PRESIDENT OF THE SOCIETY.

(*Delivered: November* 18*th*, 1904.)

IN selecting a subject for a brief address upon the opening of the fifty-fourth session of the Epidemiological Society, I have naturally turned to the field which comes more directly under my own observation in connection with factory and workshop industries. Recognising the impossibility of any adequate review of industrial epidemics and endemics in the short time at my disposal, I propose to speak mainly of a few of the broader issues, and in particular of the gaps in our present knowledge.

The conditions of indoor industrial life are in many respects favourable for the precise study of incidence of disease. There is a fairly constant population, as to which the decennial Census data are supplemented in further detail by records based upon special triennial returns furnished to the Home Office by employers. The body of workers engaged in given industries change comparatively little in average sex and age constitution, and spend many hours daily in close proximity in a common atmosphere, which in some branches of industry is that of an artificial climate, owing to exigencies of manufacture. There are, moreover, in many work-rooms exceptional conditions of environment due to the work itself, that is to say, the materials and appliances used, which may cause or predispose to disease, whether by inhalation of dust or fumes, by handling of poisonous substances, or even by direct infection.

The ordinary requirements of the Public Health Acts are

supplemented in factories and workshops by further provisions, general and special, which have done much to improve the environment, and to control the exceptional conditions which are directly or indirectly prejudicial to health. Thus a reasonable temperature must be maintained, and in general one approaching 60° Fahr. is aimed at in sedentary work; but in certain occupations the "reasonableness" has to depend to some extent upon trade exigencies, as well as upon health and comfort. In fine spinning, for example, a higher temperature is needed, and in a few branches of industry a low limit has to be allowed. Certain textile processes cannot be carried on if the air is too dry, and hence steam is added, in one way or another; but the humidity is not allowed to exceed a certain degree, and the abuses of past times in this respect are no longer found.

It is admitted on all hands that adequate ventilation is absolutely essential to health, and not less so in work-rooms than in dwelling- and sleeping-rooms. In work-rooms it is expressly required by statute, and some definition of adequacy becomes necessary. It is usual and convenient to take the proportion of carbonic acid as the index of respiratory impurity, beyond the 3 or 4 (sometimes more) parts per 10,000 found in fresh air, and various points on this scale have been suggested by different authorities of eminence as limits of permissible respiratory impurity. The only one which has statutory authority in factories at present is that which is applied to humidified cotton-cloth factories, namely, 9 parts of carbonic acid in 10,000 of air. A more stringent standard, 6 in 10,000, is to be found in the text-books; and a more lenient one of 12 is advocated by Dr. Haldane. I am not concerned here and now to criticise any of these, but desire only to point out that all are of necessity arbitrarily fixed. There is nothing to show graduated coincidence of ill-health with 6, 9, 12, or 20 parts of CO^2, or to mark in terms of disease any precise point at which increased ventilation ceases to be beneficial, or conversely any point at which a rising scale of carbonic acid begins to connote ill-health. Nor can we hope to identify such points in the future. The proportion of carbonic acid is not constant in different works, even if engaged in the same manufacture, or in different rooms of the same factory, or in different parts of the same room, or (from hour to hour) in the same part. It is affected by artificial light as well as respiration. We cannot associate given operations with precise and constant carbonic acid environment

in factories; nor are there any sickness or mortality rates ready to hand for any such groups, if they could be found. These considerations are obvious enough, and I only mention them because it has, I am afraid, been assumed in much of the recent discussion of the subject that the several standards proposed are in some degree critical points, fixed by exact experiment or experience, and that somewhere in the background are statistics to prove this.

We must be content with arbitrary standards, in ventilation as in so many other matters; and there is abundant evidence, statistical and other, to show the improvement in health which has resulted when good ventilation is substituted for bad.

Dust is being brought under fuller control, year by year, by enforcement of the general powers of the Act, and by special provision in certain dangerous processes. The result is attained in different ways: by enclosing the work so that dust cannot escape; by substitution of wet or moist processes giving off no dust; by exhaust draught carrying away the dust at the point of origin; and, as a last resource, by respirators worn by the operatives exposed to the dust. In the case of dusty lead processes in particular, great advances have been made in this direction, with resulting diminution of lead-poisoning.

Local exhaust ventilation is also of signal service where poisonous fumes are given off, but safety can sometimes be secured in another way: by finding harmless substitutes for poisonous materials, or by excluding from a given employment persons who are more susceptible by age or sex, or other reason. Thus, in some kinds of work it has been found possible to replace dangerous lead compounds by substances which are free from lead, or contain it in a less soluble and therefore less harmful form. Efficient substitutes have been found for yellow phosphorus in making lucifer matches; and the varieties of skins, hair, and wool which experience shows to involve risk of anthrax can often be replaced by others which are free from it. Children and young persons under 16 are not allowed to work in a factory without a certificate of fitness from the certifying surgeon, who has power to impose conditions as to the kind of work in which they may be employed. The employment of more susceptible persons—children, young persons, or even women—is forbidden in respect of certain processes involving risk of lead-poisoning. The employment of recently-vaccinated persons in laundries in times of small-pox prevalence is

another illustration of the same kind, though not under statute.

As regards exposure of operatives to infection, brought by fellow-workers or by materials, the Factory Act adds little to the ordinary law, beyond giving power to the Secretary of State to institute regulations, as in dangerous trades generally. Some exclusion of persons under 16 infected at the time of first seeking employment in factories is secured by the routine examination by the certifying surgeon; and the inspector can require re-examination of such persons at any time.

The age and sex conditions are usually unfavourable to the diffusion of the epidemic diseases of childhood, such as whooping-cough and measles, if introduced into a work-room. Acute cases of grave infectious disease are generally excluded by inability to work, even apart from intentional isolation; but infection of this kind, introduced in the form of slight or unrecognised attacks, or by "contacts" or otherwise, may find conditions favourable to wide transmission in the common atmosphere, shared for hours daily with numbers of susceptible persons. This consideration applies more especially to those which are most readily imparted by the breath, and doubtless the rapid spread of influenza in industrial centres is promoted by work-room congregations even more than by those of briefer duration in churches, theatres, and places of public resort. In this connection Dr. Gordon's recent Report to the Local Government Board is of great moment, and I trust that before long the Society will have an opportunity of discussing the subject generally.

Materials handled may bring with them infection, of human origin or otherwise. Clear evidence of this is to be found in the experience of laundries, notably in connection with small-pox epidemics. Rags (a larger item of import and manufacture than is generally imagined), have an unenviable but well-founded reputation as carriers of infection. Rag-sorting, paper mills, flock-making, shoddy works, all have their record of small-pox outbreaks, due to the handling of rags of unknown origin but indubitable uncleanliness; and how unclean certain classes of rags are is best known to those who have seen most of them. I can speak from experience of some which were being made into flock, and were found to lose nearly 40 per cent. of their weight on being experimentally washed. Of late years, small-pox has been less prevalent, and the use of rags in paper mills has declined; but with the revival of the

disease we may look for infection of that kind in shoddy mills and flock-making, and I believe that some indication of it has already been found in the former.

Of anthrax I need say but little after our recent discussion of Dr. Legge's admirable Paper; but it may be of interest to note that one example of infection of this kind from rags—an origin familiar abroad, but not in this country—has recently occurred in a Yorkshire mill.

The preliminary stages of three industries—wool, horsehair (including brush-making) and hides and skins—contribute practically the whole of the cases of anthrax which occur in factories and workshops. The dangerous varieties of material are in each branch fairly well understood, and where they cannot be discarded altogether special precautionary measures are directed against them.

In industrial phthisis we have to recognise something more than the spread of infection brought from outside. There is ample statistical proof, not only of relation to overcrowding and faulty ventilation, but also of relation to industries in which dust of certain kinds is breathed; and Dr. Tatham's next decennial supplement will perhaps show that the great improvements which have been made in factories and workshops in that respect have already been followed by reduction in phthisis mortality among the workpeople concerned.

I must refer only incidentally to a number of minor outbreaks of industrial disease, some of them infective in character, which attract attention from time to time: such as tetanus supervening on wounds in jute-mills, and attributed to dust from the infected Indian soil on which much of the jute is grown; the "mill fever," to which new-comers in dusty mills soon become acclimatised; the skin affections common in certain industries, such as the ulcers on the hands of fish-curers in the North, which (as Mr. Newlands, one of H. M. Inspectors of Factories, has shown) there is reason to suspect may be due to infection from diseased fish; the acute dermatitis, of which several outbreaks have occurred among men working in satin-wood. Many more examples might be given, and every year adds to the number.

Dr. Haldane and Dr. Boycott have recently established the claim of ankylostomiasis to rank as one of the infective diseases of this country: happily limited at present, as far as is known, to certain west-country mines. Research in Yorkshire mines has given negative results. The conditions which prevail in underground workings generally are

obviously favourable to the parasite if once introduced. Above ground, better arrangements can be made for sanitary accommodation; and we may hope that the disease, if it should be introduced into a factory, would fail to spread. This disease, however, will be brought before the Society at an early date by Dr. Boycott.

And now I pass on to those industrial epidemics or endemics which are due to well-ascertained inorganic poisons, and not to poisons manufactured in the system by parasitic organisms. Here the statistics are more exact, at all events as regards those (lead, mercury, arsenic, and phosphorus), which, like anthrax, are reported and investigated when they arise in factories and workshops. The field for investigation is conveniently narrowed, since we begin with a definite poison, the source of which is not in doubt, and have not to speculate upon the behaviour of organisms producing it. There is no question of isolation or disinfection, or protection by previous attack, and little of prophylaxis or acclimatisation. Nevertheless, we have to recognise a number of other factors familiar in all epidemiological studies: variations in type of attack due to the mode of presentment of the poison and the form in which it is presented, as well as dosage; varying susceptibility connected with age and sex, and individual peculiarities of the same kind; and undercurrents of unperceived attacks, shading off into dosage of even less than therapeutic proportions, which, nevertheless, if continued indefinitely, may lead in the end to organic disease.

The records of industrial lead poisoning afford examples in point. Apart from all question of quantity, lead is presented in various forms, which are far from equal in their toxic effect. The evidence goes to show that the less soluble forms—such as galena and certain insoluble silicates—are less harmful than the more soluble, which include not only ordinary white and red lead, but the monosilicate. Practical effect has been given to this distinction in applying special rules to the manufacture of earthenware and china, solubility in dilute acid roughly corresponding to gastric juice being adopted as a measure of the danger of toxic effect, so far, at all events, as concerns the lead which is swallowed. The difference is, perhaps, substantially one of dosage, after all, as it more certainly is where the small amount or intermittent use of lead, or the care taken to keep it out of the system, result in lessened danger; although it would be unwise to assume that the more insoluble lead compounds or smaller doses are altogether harmless.

We have evidence of different kinds of attack characteristic of different branches of industry, as Dr. Legge has pointed out in his published Reports. Among file-cutters, paralysis and renal changes are met with, in a degree which renders mere record of number of attacks an imperfect measure of the real gravity of the danger of the trade. A mere attack-rate or death-rate is at best a crude representation of an epidemic of any kind. The character as well as the number of attacks must be taken into account.

The remoter consequences of plumbism, such as kidney disease, may not be traced to that source, especially if work in lead has been discontinued long before death; and, if traced, may not correspond to a recorded attack. But even where death certificates are silent as to lead poisoning, they may, when massed into mortality statistics of occupation, reveal the truth indirectly in abnormal incidence of kidney disease; just as epidemics of scarlet fever have been found to be followed by increased mortality from kidney disease —obviously due to unrecorded scarlet fever—among young persons.

Similarly, in the early days of moorland water supplies, when the possibility of dangerous action on lead pipes was stoutly contested by water authorities, in spite of the appearance of lead poisoning among consumers, important corroborative evidence was afforded by the records of increased tendency to miscarriage, and even material lowering of the birth-rate.

So far as can be gathered from the official returns, the liability of women to lead poisoning is about twice that of men; and among the graver recorded symptoms, paralysis is more common in males, encephalopathy in females.

Arsenical poisoning is far less frequently reported than might have been anticipated from the number of persons who work in arsenic, and there must be a considerable degree of immunity, recalling that of the Styrian peasants, to which attention was directed years ago. There is no reason to suppose that this extends to arseniuretted hydrogen, to which the gravest reported cases are due. Nor does the evidence of influence of the manner and form in which arsenic is presented cease here, for the symptoms observed in the recent epidemics due to presence of arsenic in beer, were entirely different from those met with in either of the industrial forms. One of the results of the inquiry then made was to show that arsenic could be found in the urine and (more permanently) in the hair

of persons in whom no symptoms of poisoning had arisen. Following up this clue, Mr. W. Thompson, F.I.C., of Manchester, was led to inquire how far the common presence of arsenic in coal and coal-smoke would cause it to appear in the urine of persons living in a smoky atmosphere, and especially in the case of those whose occupations entailed special exposure to smoke. He obtained positive results in many classes of metallurgical workers—in nickel, cobalt, copper, spelter—and among town residents, as distinguished from those in the country. Arsenic is used in certain processes of glass-making, and here again positive results were obtained.

In industrial mercurial poisoning the same undercurrent is found. Definite attacks are not uncommon, having regard to the limited number of persons exposed; and side by side with them are slighter examples of the same symptoms, which would escape notice but for careful examination. In other persons, again, there are indications of presence of mercury in the system, which, though apparent on close scrutiny, are unattended with manifestations which can be called symptoms of disease. Here again there is no sharp border line; but above a certain point of dosage the presence of mercury in the system begins to be attended with interference with health amounting to poisoning. That changes, beneficial or otherwise, occur below that level cannot be doubted.

Recorded phosphorus poisoning is now happily on a small scale. Since the use of yellow phosphorus in the manufacture of lucifer matches has been brought under stricter control, and soundness of teeth secured by supervision of an official dentist, there have been but few instances of necrosis of the jaw among phosphorus workers. Attacks of this kind rarely if ever occur until the exposure has been continued for years, and this circumstance lends some support to the view that effects of the phosphorus may be predisposing rather than exciting or directly causative of the necrosis. On this view there is a gradual change, not recognisable as poisoning, or even interfering with apparent health, one result of which is to lessen the resistance of bony tissue to inflammatory processes. Attacks of necrosis have occurred after work in phosphorus has ceased.

A person working in lead, arsenic, mercury, or phosphorus is not as other men are, physiologically speaking. Bearing in mind the therapeutic use of these substances, we must admit that the change may not necessarily be harmful in every respect; but at all events clinical observa-

tion and remote mortality statistics show that it may, if the degree and duration be sufficient, sometimes lead to grave mischief, even without acute attack of severe or slight poisoning, such as is reportable to the Home Office. In short, the claims of these substances to rank among predisposing causes of remote organic disease must not be overlooked. Upon such effects official records of reported poisoning throw little light. With regard to these industrial diseases and many others, there is abundant material for study in the practice of hospitals in industrial centres, and even in local mortality records. But I would suggest to anyone who is prepared to add to our knowledge from such sources, that the more the data of disease and death can be focussed upon process and not merely trade, the more they are differentiated in terms of sex, age, duration of occupation, and character and severity of symptoms, the more valuable they become.

In the pottery industry, for example, crude statistics of illness and death due to lead or phthisis do not go far enough. There are many well-marked branches of the manufacture, carried on the same factory but involving very different degrees of exposure to dust or lead. And age and sex are so important in statistics of plumbism, and the variation in character of attack so great, that absence of differentiation on those lines would detract greatly from the value of the record. In some industries the kind of material used is an essential datum; and I can give no better example of this than the distinction drawn in Dr. Hamer's Report on Anthrax, between wet and dry hides.

As to mortality records, there is one other point to be noted. They are of necessity compiled after the industrial occupation has ceased, possibly for years, and in the absence of careful inquiry no relation to the industry at fault may appear for record. There is the further risk of a later occupation of less hygienic moment displacing in the record a more important entry of earlier work in processes injurious to health.

I have purposely dwelt upon some of the non-infective forms of industrial diseases, and upon the undercurrent of changes in the system which fall short of overt disease or poisoning, not merely because they are deserving of further study, but because they are not without suggestiveness in relation to wider questions of epidemiology; and I will turn to these for a few moments in conclusion.

Most if not all epidemic diseases present from time to

time wide variations of type, not only in individual attacks but also in character of the outbreak as a whole: shown sometimes by varying degree of intensity of attack, roughly represented by case-mortality, more rarely by predominance of particular groups of symptoms, as in successive outbreaks of influenza. In the direction of attenuation we find attacks so slight that they would pass unrecognised were not special search made. There are, further, the familiar instances in which the pathogenic organism of, say, diphtheria or enteric fever, is found, if looked for, in persons apparently healthy. Upon one view such persons are in no way affected by the infection they carry—if indeed it be infection at all—and the result has even been thought to be a reflection upon the credit of bacteriological diagnosis.

Another view is that these *pseudo* healthy persons are in truth suffering from an attack so slight in its manifestations that it is only recognisable, under present conditions of observation, by finding the specific organism, which cannot be done if the identity of that organism is not fully established.

The latter view offers a possible explanation of two epidemiological problems: first, the acclimatisation, amounting to immunity, or at all events to comparative insusceptibility without previous attack, acquired by residence in an endemic centre; and, secondly, the natural history of certain epidemic diseases in non-epidemic seasons or years. Other and adequate explanations of the latter are not wanting, more especially as regards diseases shared with lower animals, or having permanent *foci* in other countries, from which they can be re-imported. But there are epidemics which recur every few years—mumps being perhaps even a better example than measles—for which no satisfactory explanation of inter-epidemic continuity by sporadic attacks, or by re-importation from other localities, or by infection transmitted by lower animals, is always forthcoming. The possibility of persistence of infection in clothing must not be overlooked; but it can hardly be supposed that infection of mumps or measles survives long outside the body. On that hypothesis, we should have in almost every household infection ready for every susceptible person. In scarlet fever and diphtheria, occasional persistence of infectiveness after apparent recovery is now recognised.

There are other epidemics—typhus, relapsing fever, erysipelas, and, in old days, hospital gangrene—which tend to make their appearance when the conditions become

favourable, even after years of apparent absence, and in circumstances which lend no support to assumption of importation. Origin *de novo* is not now a doctrine acceptable to most of us, and we are driven back upon origin (in the sense of intensification) from something which had not, in ordinary times, betrayed its continuity of existence by causing overt disease. It is conceivable, of course, that *contagium vivum* in such a case may have maintained its existence for countless generations outside the body, ready to attack when the resistance was sufficiently lowered. The alternative view is that the outbreak may sometimes be due simply to enhanced virulence of a parasitic organism whose continuity of existence has been maintained in one human host or another, or by passing from host to host, equally in the attenuated as in the virulent phases of its cycle, with the all-important difference that in the one phase it fails to cause appreciable illness, and in the other it becomes definitely pathogenic. We know from the infectiveness of attacks so slight as to be barely distinguishable, that transmissibility is not lost when a high degree of attenuation is reached; and there seems to be no good reason to assume either that the attenuation cannot go further, or that on its doing so the organism ceases to be transmissible from host to host. And, on the other hand, the vast bacteriological flora of the alimentary and respiratory tracts afford examples of unlimited continuity of parasitic organisms which are normally non-pathogenic.

TICKS AND TICK-TRANSMITTED DISEASES.

By GEORGE H. F. NUTTALL, M.A., M.D., Ph.D., F.R.S.,
University Lecturer in Bacteriology and Preventive
Medicine, Cambridge.

(*Read: December 9th,* 1904.)

TICKS and tick-transmitted diseases promise to offer an increasing degree of interest to medical men, zoologists, and those economically concerned. Some of the diseases which ticks transmit, notably those due to the haematozoal parasites belonging to the genus Piroplasma, are amongst the most devastating which affect domesticated animals in many parts of the world. American investigators have, moreover, claimed that man is also subject to a similar disease, the so-called tick fever of the Rocky Mountains.

Before proceeding to consider some of the main points relating to tick-transmitted diseases, it appears in place to say something regarding ticks, more especially out of regard for those who have perhaps no special knowledge of these ectoparasites.

TICKS.

Ticks belong to the class Arachnoidea, in which are also included the spiders and mites; all the members of this class possessing eight legs when adults. Ticks constitute a well-defined group or family, the Ixodidae, in the Order Acarina. Neumann, in common with most zoologists, divides the Ixodidae into two sub-families, the Argasinae (including the genera Argas and Ornithodoros) and Ixodinae (including the genera Ixodes, Eschatocephalus, Aponomma, Amblyomma, Hyalomma, Haemaphysalis, Rhipicephalus, and Dermacentor). It would be out of place here to dwell upon the particular characters possessed by the different genera above-mentioned, especially as these can be found described in special works relating to the subject. (See Neumann, 1896-1901, and Salmon and Stiles, 1902.)

We are indebted to a long list of zoologists for what we know of ticks but our knowledge in the main refers

to the external features of tick anatomy. In the case of mosquitoes, the study of the diseases they transmit led to a more thorough investigation of their biology and internal structure, especially at the hands of medical men; and it is safe to say that the same impulse is leading us to turn our attention more closely to ticks.

Ticks, as far as known, derive their nourishment entirely by sucking the blood of their hosts. These hosts are almost exclusively terrestrial vertebrates, and include practically all classes of Mammalia, Aves, Reptilia (Chelonia, Sauria, Ophidia), and Amphibia (Bufo). Neumann records but two species as attacking insects (Coleoptera).

The geographical distribution of ticks is world-wide, but they are most numerous in warm countries, where they naturally find the conditions of temperature and moisture which are most favourable to their metamorphoses on or near the surface of the ground. The influence of low temperatures and desiccation in retarding, or completely checking, the developement of ticks from the egg to the larval and subsequent stages, has been already demonstrated in the laboratory. Ticks follow the lines of travel of their hosts. Some ticks attack a variety of hosts, whilst others appear to be more selective in this respect, and to be more limited in their geographical distribution.

The structure of ticks shows evidence of adaptation to a life of parasitism. The legs terminate in sharp claws and pads (the latter absent in Argasinae), with which they cling with wonderful tenacity to passing objects and animals. The pulvillum or pad permits even the ponderous gorged female tick to climb up vertical glass surfaces. The rostrum, with its beautiful structure, serves as a boring organ; the recurved teeth of the hypostome and the sharp denticles of the mandibles affording the tick a firm hold in the skin of its host, to which it may remain attached for a brief period (Argas), or for days.

Commencing with the egg, which is laid in recesses in the ground, the tick begins its chequered career, together with hundreds or, indeed, thousands of its fellows laid by its fertile mother. The period of metamorphosis, which ends in the hatching of the six-legged larva from the egg, is much influenced by temperature, being prolonged for months in cold weather. The larva creeps out, and, its chitin having hardened, it crawls upon the surrounding vegetation, there to play a wonderful game of patience, which may last for weeks or months, until it is disturbed into amazing activity by some passing animal, to which it promptly clings

as it brushes by through the tick-infested grass or shrubbery. The larva now seeks a suitable place upon the skin of its host, and in a few minutes may be seen boring its rostrum into the skin, and soon after it begins to suck the blood of its host. In some ticks, the larvæ having gorged, drop off upon the ground, there to undergo their metamorphosis into eight-legged nymphs, which in turn attack a fresh host in the manner described for the larvæ. This is the case, for instance, in *Haemaphysalis leachi*, which transmits canine piroplasmosis. In other ticks, the metamorphosis from larva to nymph may take place upon the host; this, for example, being the case with *Rhipicephalus annulatus*, which carries Texas fever. The nymph having gorged, may or may not drop off to undergo its metamorphosis upon the ground, emerging as a sexually mature tick. In the dog-tick just mentioned, then, the larva, nymph, and adult each drop off in turn when gorged, so that a single tick, to reach maturity, may be obliged to find and attack three different hosts. In view of the great loss of life this must entail in the ticks which fail to find a host, even after months of waiting, we can readily understand why a parent tick lays several thousand eggs. Starting with as many larvæ as there were eggs, it is certain in many cases that but few individuals survive to the adult stage, owing to the many vicissitudes to which they may be exposed.

The Effects of Tick Bites.

In a paper published in 1899, I described some of the effects of tick bites, and the accidents which may follow the attacks of various species of ticks on man and animals. Where an animal is attacked by large numbers of ticks, the cutaneous irritation and the loss of blood undoubtedly cause considerable injury. The seat of the bite is inflamed, and may serve as a point of entrance for infective agents. When ticks are forcibly removed, the rostrum may remain anchored in the skin and lead to serious consequences, which appear in some cases to depend upon infection from without; in others, possibly upon the toxic action of the embedded rostrum, with its attached soft structures. In other cases, toxic local or general effects have followed tick bites in a very brief period of time, suggesting the injection on the part of the tick of some poisonous substance into the wound. Some have attributed these effects to a toxic saliva, but it appears to me possibly due to disgorged

food residue. In any case, experimental investigation is needed to explain the cause of these effects. Cases are recorded in the literature of ticks boring themselves completely beneath the skin, and, in some instances, giving rise to the formation of abscesses, etc.; these seem to be exceptional occurrences.

Ticks as Disease Carriers.

There are several classes of diseases which are known at present to be transmitted by ticks. The diseases, which may be conveniently grouped under the class Piroplasmosis, being named according to the genus of parasites which cause them, affect a number of Mammalia, the protozoal parasites being apparently peculiar to each species of host. A disease of the domesticated fowl, analogous to relapsing fever in man, and likewise due to a spirochæte, has been proved to be transmitted through the agency of a tick. Heartwater, a disease affecting sheep and goats in South Africa, has also been communicated to these ungulates through the agency of infected ticks. In all these cases we have clear experimental evidence to go on.

The part played by the notorious *Argas persicus* in relation to a fever which occurs in Persia, and has long been attributed to the bite of this species of tick, also the grave effects attributed in Africa to the bites of *Ornithodoros savignyi* (Audouin), still require investigation. In both instances these ticks attack man. Further evidence is required with regard to the supposed part played by *Dermacentor andersoni* (Stiles, 1904) in the transmission of the disease known as Tick Fever of the Rocky Mountains. Reasoning by analogy, it is highly probable that species of tick will be found in the future to be the carriers of different species of Piroplasma. The discoveries in connection with the fowl disease above referred to are highly suggestive in their bearing upon the probable mode of transmission of relapsing fever to man. Although "louping ill" of sheep has been attributed to the consequences of the attacks of ticks, no positive evidence, worthy of serious consideration, has as yet been gathered in this respect.

In the Table on following page I have briefly summarised certain facts regarding tick-transmitted diseases.

When we come to consider the stage in its development at which a tick may transmit parasites, we find that this varies according to the species of parasite and species of tick concerned. In the diseases due to Piroplasmata, the

mother tick, having gorged on infected blood, drops to the ground in the usual manner, and in due course lays her eggs. These eggs are infected, and from them issue infected

Name of the Disease.	Animal Affected.	Geographical Distribution.	Tick Concerned.	Parasite Transmitted.
Texas Fever, or Redwater	Cattle ...	N. and S. America	Rhip. annulatus	Piroplasma bigeminum
		Africa, Queensland	Rhip. australis	"
		Asia, Europe .	Ixodes reduvius ?	"
Rhodesian Fever	Cattle ...	Africa ...	Rhip. appendiculatus	Piroplasma parvum
Carceag ...	Sheep ...	Europe ...	Rhip. bursa ...	Piroplasma ovis
Canine Piroplasmosis	Dog ...	S. Africa ...	Haemaphysalis leachi	Piroplasma canis
		Europe ...	Ixodes reduvius ? Dermacentor reticulatus	
Piroplasmosis .	Horse, Donkey, Mule	S. Africa ...	?	Piroplasma equi
		Madagascar ...	?	"
		Venezuela ...	?	"
		Germany, Italy	?	"
Piroplasmosis .	Monkey ...	Uganda ...	?	Piroplasma nov. spec.*
Rocky Mountain Fever: Piroplasmosis ?	Man ...	Western U.S.A.	Dermacentor andersoni ?	—
Heartwater ...	Goat, Sheep	S. Africa ...	Amblyomma hebraeum (Koch)	Unknown
Spirochæte Disease of Marchoux & Salimbeni	Fowl ...	Brazil ...	Argas miniatus	Spirochæte Marchouxi

* Unpublished. See Paper by Philip Ross in forthcoming Number (January, 1905) of *Journal of Hygiene.*

larvæ ; but these do not necessarily convey the parasite in their larval stage of existence ; for instance, in the case of:—

Rhipicephalus annulatus (Say) and *Rhip. australis* (Fuller). The larval tick is able to transmit piroplasmosis to cattle.

Rhipicephalus bursa (Canestrini and Fanzago). Larva and nymph innocuous, adult infective to sheep, to which it communicates piroplasmosis.

Haemaphysalis leachi (Audouin). Larva and nymph innocuous, adult infective to dogs, to which it communicates piroplasmosis.

On the other hand, according to experiments cited by Theiler (1904), and work by Lounsbury:—

Rhipicephalus appendiculatus (Neumann). Fed as a nymph on cattle suffering from Rhodesian Fever, communicates the disease as an adult tick. If infected as a larva, it is infective as a nymph.

Again, in the case of *Amblyomma hebraeum* (Koch), which conveys the unknown parasite, which is the cause of Heartwater, Lounsbury (1900) states, that when the tick is fed upon an infected animal in the nymph stage, it communicates the disease as an adult. Or when fed in the larval stage, it proves infective as a nymph. In other words, as Mr. Lounsbury informs me, both in the case of Heartwater and Rhodesian Fever, the parasites are conveyed by the ticks—as he expresses it—"stage by stage." Nothing is known regarding the life-history of the parasites above mentioned within the bodies of the ticks.

On the other hand, in the case of the spirochætes of the fowl, these protozoa appear to multiply in *Argas miniatus*. When in Paris some months since, Dr. Borrel, of the Pasteur Institute, informed me that the spirochætes, after entering the stomach of the tick, together with the blood from the diseased bird, wander out through the stomach wall of the tick, and are found swimming about in the tick's blood; in a similar manner to what they do in the blood of the vertebrate host. The *Argas* does not become infected, and consequently infective, subsequent to feeding on spirochæte containing blood, unless it is maintained at a suitable temperature, say of 30 deg. C. As Dr. Borrel showed me in Paris, it is easy enough to demonstrate if a tick is infected or not. This is accomplished by snipping off the extremity of one of its legs, and examining the drop of blood which escapes for spirochætes.

Persistence of the Parasites in Ticks.

Once infected, *Argas miniatus* is capable of harbouring the parasite for a considerable length of time, and consequently of communicating the disease to a healthy fowl five or more months after it has ingested spirochæte containing blood. The mechanism whereby the spirochæte issues from these ticks into the vertebrate host is as yet unknown.

Observations on ticks harbouring Piroplasmata show that the latter may persist for months in the tick. For instance, in *Haemaphysalis leachi*, the *Piroplasma*

canis continues not only to live through all the stages of development of the tick from egg to adult, as Lounsbury showed—and this may be for many months—but it persists a long time in the adult tick. Working in Cambridge with adult ticks sent me by Mr. Lounsbury, I found them still capable of setting up a fatal infection in a dog, after they had been kept unfed, as adults for a period of seven months at room-temperature. As a number of these adult ticks died during these months, and the survivors proved infective, it is reasonable to conclude that the Piroplasma only died with the tick. Redwater can be communicated by larval ticks which have starved for months.

Incubative Period.

In all the above tick-transmitted diseases, the attack of the tick is succeeded by a period of incubation of several days. Experiments made in the laboratory with infected ticks have shown results comparable to those observed in nature. They are, however, necessarily more accurate.

Disease.	Incubation in Days.	Observer.
Texas Fever	10	Smith and Kilborne (1893).
Rhodesian Fever ...	8	Theiler (1904).
Tristeza	12 to 28	Lignières (1900).
Sheep piroplasmosis ...	5 to 6	Motas (1904), fever and parasites on tenth day.
Canine piroplasmosis ...	13 to 21	Lounsbury and Nuttall (1901 to 1904).
Heartwater	11 to 35	Lounsbury (1900).
Fowl Spirochæte Disease .	4 to 7	Marchoux and Salimbeni (1903).

The Piroplasmoses.

The diseases affecting cattle, sheep, deer, equines, dogs, and apparently monkeys, and possibly man, are grouped together as piroplasmoses, for the reason that they are caused by parasites, which at present are classed under the genus Piroplasma. The Piroplasmata are Protozoa, and, as their generic name implies, resemble each other morphologically in that they are pear-shaped, micro-organisms, occurring singly or in pairs, or more or less even-numbered groups of four to sixteen individuals within the infected animals' red blood corpuscles. Besides the typical pear-shaped organisms, spherical forms may be encountered, which resemble young forms of human malarial parasites. Working with *Piroplasma canis* in Cambridge, Dr. Graham-

Smith and I have observed large sausage-shaped extracellular parasites occasionally. These forms offer a certain resemblance to our malarial crescents, and it appears likely to me that they represent gametes. These forms have not as yet been described. The intracorpuscular piroplasmata, at certain stages of their development, apparently before they take the pear shape, may show active amœboid motion. Stained by modifications of the Romanowsky method, the protoplasm stains blue, the nucleus red. The protoplasm may show vacuolisation. In extravascular blood, Lignières described the occurrence of flagellate bodies in bovine piroplasmosis, "la Tristeza," of South America. Bowhill has since observed similar bodies in connection with other species of piroplasmata, and has been the first to photograph them. The significance of these bodies requires explanation, although from what we know of human malarial parasites, it seems tempting to make comparisons.

The parasites which occur in the horse and in Rhodesian fever appear to me possibly different to the others. Injection of large quantities of blood containing these parasites into susceptible animals appears, as a rule, to be without effect. The parasite of Rhodesian fever behaves differently with regard to ticks than do the others. The parasite is, moreover, different in its morphology, as we shall presently see. With the exception, then, of the equine and the Rhodesian-fever parasites, and the parasites affecting the deer, man, and monkey, about which we want more knowledge, we find that infection is readily set up by inoculation of blood containing the piroplasmata, affecting cattle, sheep, and dogs in different parts of the world.

If we, moreover, compare the last-named diseases with regard to their symptomatology and pathology, we are at once struck by their great similarity. In all we have the disease ushered in by fever, often preceded by the appearance of the piroplasmata in the blood. There is a great destruction usually of red-blood corpuscles, and the serum is tinged with hæmoglobin, which finds its way into the urine. Hæmoglobinuria is a usual, but not invariable, symptom. This also holds for the icterus. The animals show loss of appetite, prostration, accelerated breathing; the mucous membranes are pale; emaciation is especially marked in chronic cases. A fall of temperature far below normal precedes death. At autopsy the blood is found to be thin and watery, the serum tinged with hæmoglobin; the spleen and liver are often much enlarged and congested, etc. The gross pathological lesions in the organs may, how-

ever, be very slight. The blood count may show a reduction in the number of red-blood corpuscles to one-third, or less than the normal.

Immunity follows recovery; but the term immunity must be understood in a relative sense only, for the reason that the parasites, although not demonstrable microscopically, may persist for a long time in the blood of animals which have outwardly everyappearance of health. That the parasites are present in recovered animals can be demonstrated by inoculation of their blood into susceptible animals. In this manner, Schroeder, in the United States, was able to demonstrate that the parasites of Texas fever persisted in the blood of recovered animals, so-called immune animals, for six to eight years. In other words, the condition is one of latent infection. That this is the case has also been shown in Turkey, South Africa, and the Transcaucasus, where attacks of Rinderpest have been seen to lead to a recrudescence of the piroplasmosis in animals known to be "immune" to Redwater. Similarly, exposure to cold, fatigue, or other inimical conditions may lead to a recrudescence of the disease after long intervals of apparent health. Quite recently it has been observed in the Philippines, by Jobbling and Woolley (1904) that apparently healthy cattle, imported from China, harboured piroplasmata, for inoculations with their blood into healthy cattle imported from the northern United States resulted in the latter developing piroplasmosis. Similarly, in canine piroplasmosis, Robertson (1902) has found the blood of dogs infective up to four and a-half months after recovery. The significance of this persistence of the parasites will be the more appreciated when I add that the "recovered" animals may serve as fresh centres of infection of the ordinary carrying agent of the disease, the tick, and this for an indefinite time.

Epidemiology of the Piroplasmoses.

It has been observed in connection with Redwater, canine piroplasmosis, etc., that the disease is one of locality. Cattle are prone to acquire the disease in certain low-lying pasture lands, the dangerous area being at times quite clearly marked by the fence which limits a given field. Stabled cattle escape the disease. Dogs which are taken out hunting in certain districts acquire the disease, whilst those kept in town escape. The piroplasmoses occur especially in the spring and summer, this corresponding to the period when ticks are found upon their hosts. Animals indi-

genous to the soil are relatively resistant, this being doubtless due to their having grown up continuously infected from youth. The behaviour of the piroplasmoses in this respect reminds us of what has been observed in human malaria. Calves are more resistant than adult animals; in fact, as age advances, cattle appear to become more susceptible, for the disease is frequently observed in old cows living in endemic areas. In the case of the canine malady, on the other hand, the disease is more fatal to puppies than to adult dogs.

The influence of locality, season, etc., on the prevalence of these diseases is readily understood when we consider the biology of the ticks which carry the infectious agents.

Rhodesian Fever of Cattle.

The bovine piroplasmosis known as Rhodesian or East Coast Fever of South Africa is possibly identical with the "tropical piroplasmosis" of cattle observed in Transcaucasia this year by Dschunkowsky and Luhs. The parasites differ from those encountered in Texas fever or Redwater, and the pathology of the disease is not the same. Cattle immune to Texas fever are susceptible to Rhodesian fever. The parasites of Rhodesian fever are smaller than those described in Redwater, and Theiler (1904) considers them a different species, to which he gives the name of *Piroplasma parvum*. Whereas Redwater is readily communicated to healthy animals by inoculation with blood containing *P. bigeminum*, corresponding experiments with blood containing *P. parvum* have proved negative, even when injections of huge quantities of infected blood were practised (Theiler, Dschunkowsky and Luhs). These facts alone indicate that we are dealing with distinct diseases which are set up by different parasites. It is, moreover, highly probable that what we call *P. bigeminum* to-day is actually a collective term for several parasites belonging to different species. The determination of these species may not be possible on morphological grounds alone; we have to call in aid the species of tick which transmit them, as also the test of immunity in animals. It is not at all impossible, it appears to me, that a similar view will have to be held in the future with regard to human malarial parasites, which have hitherto been regarded as single species.

Returning to *P. parvum* (Theiler), I would state that it occurs in spherical, bacillary, and intermediate forms, and

that it reacts like the other species of Piroplasma to Romanowsky's stain. The disease is especially fatal; Koch (1904), for instance, stating that the loss in infected herds commonly amounts to 90 per cent.

Mixed Infections.

As Theiler points out, mixed infections with *P. bigeminum* and *P. parvum* may occur, a fact which has certainly led to both species having formerly been considered one by Koch and Laveran. There is further reason for such confusion in the statement made by Theiler that the inoculation of *P. bigeminum*, contained in the blood of recovered cattle, into calves, gives rise to the appearance in their blood of parasites resembling *P. parvum*, alongside of the typical *P. bigeminum*. Moreover, the latter parasites in the blood of salted cattle resemble *P. parvum*. This peculiar appearance of *P. bigeminum* would seem to depend upon inimical conditions operative in the bodies of salted animals.

I have already referred to the co-existence of Rinderpest and Piroplasmosis in cattle. Animals may harbour Trypanosomata and Piroplasmata, or the latter and Spirochætes. Similarly in horses, we may find mixed infections. Thus, in a specimen of blood recently photographed by Mr. T. Bowhill, at the Cape, a picture of which I shall project upon the screen, you will see *Piroplasma equi* present, together with embryo Filariæ, and this in a horse that died of horse-sickness! It will be readily understood how confusing such observations are, and how easily an investigator may fall into error regarding the actual cause of a disease.

Spirochæte Disease of Fowls.

The disease of fowls observed by Marchoux and Salimbeni (1903) in Brazil, and which they succeeded in transmitting by means of infected *Argas miniatus*, is one of considerable theoretical importance. The disease is usually very fatal and acute. The affected birds suffer from diarrhœa, anorexia, somnolence; the comb is pale, the birds lie down and die in convulsions. The disease may be subacute, lasting eight to fifteen days, during which the bird becomes emaciated, and paralysis appearing first in the legs, extends to the wings. Recovery is rare, either in the acute or chronic form. Fever appears at the onset, and lasts four to five days (42-43 deg. Cent.), then it falls to below normal before death. When recovery takes place, the temperature gradually returns to normal.

At autopsy, in acute cases, the liver is found greatly enlarged, fatty, and showing focal necroses. The other organs appear normal. The blood is claret-coloured and fluid. In chronic cases all the organs, even the liver and spleen, appear atrophied.

The spirochætes multiply up to the death of the animal, finally occurring in agglomerations and in great numbers in the blood. Corresponding with the crisis which ushers in the commencement of recovery the parasites disappear, owing to causes apparently similar to those which operate in relapsing fever in man. The spirochætes resemble *Sp. obermeieri*, and little is known about them. Their position amongst the Protozoa is as yet undetermined. Suggestive in this connection is the recent work of Schaudinn (1904), who has described a spirochæte-like stage developing out of a Trypanosoma, which represents in turn a stage in the life-history of an endoglobular parasite of birds, the Halteridium.

Contrary to what has been observed hitherto in the Piroplasmoses, the spirochætes appear to be less selective with regard to the species of host. For instance, human relapsing fever has been communicated to monkeys (Cercopithecidae); and the avian spirochætes here referred to possesses a fairly-wide range of pathogenicity amongst birds other than fowls. Thus, Marchoux and Salimbeni, and Levaditi (1904) more recently, have been able to infect the goose, duck, turtle-dove, pigeon, sparrow, lark, etc., with the new spirochæte; I say "new," for the reason that it appears to be different, in pathogenicity at any rate, from *Sp. anserini* of Sakharow. Nevertheless, this apparent wider range of pathogenicity amongst birds may receive a partial explanation in the fact that birds belong to a group of relatively closely-related vertebrates.

Contrary, again, to what has been observed in Piroplasmosis, there is no evidence that the spirochætes persist in the body after recovery; moreover, the immunity acquired after an attack is but of brief duration.

IMMUNISATION.

The subject of artifically-induced immunity has naturally engaged the attention of many workers, more especially with regard to the piroplasmoses of cattle, dogs, and sheep. It was already observed by Smith and Kilborne (1893), with regard to Texas fever, that recovered animals remain protected in a large measure in succeeding years, and that

animals which have suffered from the disease when young appear to remain protected longest. They found that animals inoculated with Texas fever blood in winter acquired a milder form of the disease, and remained protected. Since then an extensive experience has been gained in different parts of the world, cattle receiving inoculations of blood from animals suffering from mild Redwater, or the blood of "salted" cattle (preferably calves), being used for preventive inoculation. The loss from these inoculations may vary from 3 to 25 per cent.; but this is small compared to, perhaps, the complete wiping-out of unprotected herds in which the disease may appear. Whereas this mode of preventive treatment seems justified in regions where the disease is endemic; it seems scarcely justified otherwise, in view of the fact that "salted" animals are a source of danger for years, wherever they go if accompanied by suitable ticks, which can convey the parasite to healthy animals.

In the case of Rhodesian fever, immunity has not been produced artificially with success of a practical character. Koch (1904) recommended repeated inoculations with recovered blood; but experiments conducted on a scale of 5000 animals have been deemed sufficient evidence of the method being a failure by the veterinary authorities in South Africa (Gray, VII., 1904; Theiler, XI., 1904).

Finally, treatment with the serum of immune animals cannot be said to have afforded results promising to have any practical value. Nevertheless, it may be noted that Nocard and Motas considered that inoculations with the serum of hyper-immunised animals afforded protection in canine piroplasmosis. More recently, Motas (1904) has carried out corresponding experiments with sheep piroplasmosis; but here again the results do not seem promising from a practical standpoint. It is impossible here to enter more fully into the problems relating to immunity in these diseases.

It would lead me too far afield to fully discuss the work that has been done on immunity in connection with spirochæte infections. Confining myself to the spirochæte disease of fowls referred to previously, I would state that Marchoux and Salimbeni report that infected serum heated five minutes at 55 deg. C., or infected serum kept two to four days at room-temperature, is not infective but protective. Fowls immunified therewith yield a protective serum. They also protected animals by inoculating them with a mixture of immune serum and blood containing

virulent spirochætes. Immune serum proved of no value therapeutically. Levaditi has, however, claimed since, that immune serum may hasten the crisis. Immune serum agglutinates and immobilises the spirochætes.

Prophylaxis.

Apart from necessary attempts to produce immunity in animals exposed to infection, there are several measures of prevention which have been recommended and actually resorted to in connection with piroplasmosis of cattle. Morgan (1898) advises that infected pastures shall not be used for grazing cattle on for a year. He gave this advice for the reason that cattle placed on such a field after it had been used for pasturing horses and mules for two years, did not develop the Texas fever. Lignières (1900), from his South American experience, recommends the sowing of clover on pastures. It appears possible that the changed vegetation may exert an influence, and the experiment is worthy of trial in suitable cases. The fencing-in and drainage of infected land has caused the disease to disappear in parts of Finland. Quarantine measures have been extensively adopted in the United States, in view of long experience proving that cattle driven northward from the endemic areas carried the infection with them. The disuse of fields, as recommended by Morgan, has an obvious scientific basis, for the ticks will die out in the absence of suitable hosts; or if they survive by attaching themselves to other hosts, they will, with time, cease to harbour the parasite they transmit. Destruction of ticks by burning of the grass on infected pastures in the autumn has also been advocated. Finally, dipping cattle has been resorted to on an extensive scale. In the latter case the cattle are driven through tanks, in which they may be obliged to swim in a bath containing constituents such as mineral oil, glycerine, sulphur, arsenic, etc. The process may have to be repeated several times. Unfortunately, the dip has its disadvantages; a certain number of animals are injured, and ticks adhering about the head are not affected. Nevertheless dips necessarily serve a useful purpose in lowering the number of ticks on animals.

Note Regarding the Geographical Distribution of the Piroplasmoses.

I have attempted, in the accompanying map, to give a general idea of the wide distribution of the piroplasmoses in different parts of the world. Most of the blackened areas

refer to bovine piroplasmoses. The distribution of the forms of piroplasmosis affecting different animals, exclusive of countries where diseases having similar symptoms occur, is about as follows :—

Bovine piroplasmosis (Redwater, Texas fever) occurs in the United States, West Indies, Southern Venezuela, Uruguay, Paraguay, Brazil, Queensland, various parts of Africa and Europe, India, China, the Philippines.

Rhodesian fever occurs in Rhodesia and the South-East Coast regions of Africa ; apparently also in Transcaucasus.

Ovine piroplasmosis occurs in Roumania, Italy, France, West Indies, the United States, South Africa.

Equine piroplasmosis occurs in South Africa, Madagascar, Germany, Venezuela.

Canine piroplasmosis occurs in Italy, France, North and South Africa, India.

Discussion on Dr. Nuttall's Paper.

Sir Patrick Manson : I have no doubt, gentlemen, that you will concur with me in expressing an appreciation of the exceedingly interesting, and, I might say, important paper which we have listened to, and the beautiful demonstration that Dr. Nuttall has given us.

Before I was invited to occupy the dignified position of presiding on this occasion, I was asked to open a discussion on Dr. Nuttall's paper. I suppose the fact of my promotion will not invalidate that arrangement: at any rate, I shall assume it does not.

There are many points in Dr. Nuttall's paper that suggest discussion, more particularly, to my mind, the bearing of the facts he has adduced on human pathology. I have noted a few points, and although I will not venture to discuss them all, there are one or two that I should like to mention. What struck me first was that the symptoms of piroplasmosis are exceedingly like those of that most important disease affecting man in hot climates, viz., blackwater fever. The occurrence of hæmoglobinuria and icterus, which are almost characteristic of piroplasmic diseases, are also the two most characteristic and important symptoms in blackwater fever. This is usually put down as a malarial disease, and there is a good deal of evidence in favour of such a supposition. But there is also a certain body of evidence tending to show that, even though it be malarial, this infection is not the only cause, or rather is not the entire cause of blackwater

fever: it may be a determining cause of the explosion and manifestation of the disease, but it is not the essential microbic cause of the malady.

Dr. Nuttall mentioned just now the fact (now generally recognised) that animals infected with piroplasma, yet apparently immune from symptoms, can be induced to manifest the characteristic pathological effects of piroplasma infection by inoculation with some other micro-organism, by exposure to cold, or by any other similar strain upon the physical powers. Exactly the same thing has been noticed in the case of blackwater fever, suggesting the hypothesis that this, too, is a tick infection which for a long time remains latent, until some intercurrent circumstance, such as a malarial attack, an overdose of quinine or exposure to cold, sets the latent parasite off, so to speak, on its pathogenic course. That idea has long been present to my mind, and Dr. Nuttall's remarks have accentuated it.

An excellent object of investigation, then, would be the hypothetical piroplasma of blackwater fever and the tick, or whatever intermediary it may be, that transmits the disease.

I was rather disappointed in one part of Dr. Nuttall's paper: I had hoped that he would give us some information about a curious disease that they get along the course of the Zambesi, and which is certainly produced by a tick, known as the Garrapato disease, and was first noticed by Dr. Livingstone; but it has since then been observed by many others —Sir John Kirk and Dr. Daniels, for example.

Looking over some papers, two or three days ago, I came across a letter from one of my old students, Dr. A. Park Ross, at the School of Tropical Medicine, who has recently gone out on a Boundary Commission to the Zambesi region. When I heard that he was going there, I requested him to make some enquiries on this particular subject—Garrapato disease. This is his letter; it is of so much importance that I take the liberty of reading it.

"River Loangwa, Lat. 15° 5′, 25th August, 1904."

"I ought to tell you that opportunities of doing medical or scientific work here have been few, and the following is as far as I have got with the Kufu disease (Kufu is the native name for Garrapato). The latter is not nearly so common as I was led to expect at Tete, and the reasons for the belief in its almost universal prevalence in this country are—first: that Kufu (that is, the tick) occurs in almost every dirty village (and these are many); and, secondly: that the natives ascribe almost every serious disease to it, in consequence of which I have had several wild-goose chases after it.

"The following clinical description of the disease, is taken from the statements of four exceptionally intelligent natives, each from a different part of the country, and each of whom has had the disease.

All their statements tallied. I required no interpreter, and took care to ask no leading questions.

"The disease extends from Angoni Land westwards, at least as far as the confines of North-western Rhodesia. It shows no preference for high or low ground, as it occurs in the Tanganyika Plateau, 5000 ft. above sea level, and on this river 1000 ft.: it occurs also at Tete, 340 ft. above sea level. It occurs during the rains, December to March; but is much more prevalent during the dry season. I could elicit no information as to its occurrence in epidemic form. Cases occur sometimes among the Kufu-infected villages, but the great majority occur in strangers sleeping in such villages, whose own villages are not infected. On the other hand, many of the villages infected with Kufu do not report the sickness, and the natives invariably say that sometimes bites produce sickness and sometimes not. It is impossible to get at the infant mortality in such a village, and the information as to whether Kufu is a factor would be unreliable if derived from any other source than a European doctor. Curiously, the idea was suggested by a native that a universal infection in childhood in such villages was the cause of the relative immunity of adults. Natives living always in infected villages do not consider themselves likely to acquire the disease, whereas natives whose own villages are not infected are very careful as to sleeping at huts in an infected village.

"The Kufu inhabits the dry earth in native huts, and can be found during the day about two inches below the ground, generally close to a hut-post or bed-post. It does not bite during the day, or where there is light (as I have proved myself).

"The following account was given by our interpreter, an intelligent man, who had had the disease, and it was corroborated by three of our personal servants, who also had had it. The bite produces irritation for two or three days, during which the patient otherwise feels well. On the third or fourth day fever begins: there may or may not be a rigor, but there is always violent headache and severe pain in the small of the back, and in the bones and joints generally. None of these boys admitted gastric pain, but one said he felt inclined to vomit. There is copious waterish diarrhœa, of a very dark colour, and the urine is also dark. (I would not like to pin my faith to the last statement, but three of the four were positive upon the point.) The pain in the back disappears when once the diarrhœa is established. This condition lasts for about two weeks, after which recovery is complete. I can elicit no history, and can see no signs of sequelæ. One attack confers immunity. A pregnant woman may, and generally does, abort during an attack. The disease is much more severe in children and in old people.

"Now, as to what I have seen myself. I first got on the track of it at Misah, when it was reported at Fort Young, 25 miles distant; but, on arriving there, I found the infected huts burned, and the natives could not, or would not, show me a case. At Fort Jameson, Dr. Cole showed me two cases sent in as Kufu, which were really suffering from another disease.

"I have twice tried to inoculate myself, but each time without result. I got three ticks in a house in Fort Jameson, which natives said was recently infected. I fed them on blood, but only one would bite: the other two refused to bite, and died. At Fundas village there were Kufu in every house. None of the people were ill, but a stranger coming there two weeks before had the disease, and had been carried away to his own village, where I could not follow him. I took four active ticks from this house, and put them on my arm in order that

they might bite. I found that when exposed to the sun they would not do so, but began at once when a cloth was put on them. The pain of the bites was not very perceptible, and I should have doubted their activity had not each tick swelled up like a balloon. When collectively they had withdrawn about half a c.c. of blood, I separated them with tobacco smoke: the bites were indicated by purple patches due to extravasated blood under the skin, and enough blood to make a film exuded from one of them. I covered the bites with a soft bandage: there never was the slightest irritation, and in three days the blotches disappeared. In seven days a little pink papule appeared over each bite; this lasted for several days and then disappeared. A fortnight has now passed, and I have never felt any symptoms of any kind.

"I was informed in Tete that, some years ago, tick fever was a serious deterrent to the slave trade. Dr. Livingstone's statement as to death occasionally occurring, I had corroborated by natives and in Tete; but the former allege that the disease is not so common as in the days of their forefathers. There are not so many villages burned for it. I have only seen two in some 4,500 square miles of country. An old man said it was not always in the country, and came from the north."

This Garrapato disease is certainly a subject well worthy of investigation, and I hope that anyone who has an opportunity of studying it will not fail to take advantage of it.

One word before I close these observations. What struck me about Dr. Nuttall's paper — and, indeed, about the entire subject of tick diseases and protozoa diseases of man and the lower animals—is the enormous extent of the subject and the rapid growth that it is undergoing. This fact we have recognised for some time at the London School of Tropical Medicine; and with the view both of giving instruction to our students and of advancing the study of the subject, we have determined to institute two Chairs, one dealing with protozoa and another dealing with Helminths. We hope to still further advance the subject in that way, and prepare men who may be of value as investigators. This, I think, is a step in the proper direction; but I think the subject is so extensive and important that the medical profession generally should recognise it more definitely than they do at present. I think Chairs of, say, Medical Zoology and Medical Haemology ought to be instituted in our great Universities, more particularly in the University of London. I think it would be a good thing if this Society could give expression to some idea of that sort.

DR. THEODORE THOMSON: It is very kind of you to call upon me, but my sole acquaintance with ticks has been a strictly personal one, and I regret to state has not been so painless and so little disturbing as that of your friend in

Africa. I have practically no knowledge as to ticks, and therefore I can say nothing about the subject.

DR. BENTLEY: I have had very little experience of tick-produced diseases. I have, however, met with two species of piroplasmosis in dogs in Assam, one of which showed a very small parasite, occurring sometimes in couples and sometimes in threes and fours in a corpuscle, apparently answering to the description usually given of the piroplasmon in cattle. Another kind was much larger, about three μ in diameter. The first variety I mentioned I saw in an acute attack that took place in a dog that had been brought from England the previous year. This dog was brought to my bungalow by the man who owned it, and who had noticed that it was suffering from severe anæmia, and, apparently, symptoms of syncope. I took its temperature, and found that it was suffering from fever at the time: on making blood-films I found an enormous number of small parasites, which occupied nearly every one of the corpuscles. The dog died in three days; it suffered a good deal, and showed signs of icterus and hæmoglobinuria. It struck me at the time that the symptoms of this case very much resembled those of blackwater fever which one sees described in text-books. That is all I can add to the discussion.

PROFESSOR W. J. SIMPSON: I am afraid I cannot make any remarks on this interesting question, further than to say that it seems to me rather alarming that the inoculation should spread the disease rather than prevent it, especially in the case of this Rhodesian fever. It is quite evident that some other kind of treatment will be required; and it would seem that it should rather be sought for in something similar to the quinine which is used for malarial fever rather than in inoculation, which is so effectual in other diseases.

I have listened with great pleasure and also with very great benefit to a most interesting lecture, and I thank Dr. Nuttall very much for it.

DR. G. C. LOW: I have listened with much pleasure to Dr. Nuttall's paper. This subject is now becoming of much more importance, both from the animal point of view and from the human point of view also. We have seen an enormous destruction of cattle and other animals brought about by tick diseases: and we know that much valuable

work has been done in America in connection with this subject in recent years.

I have heard recently from the principal medical officer of Natal, who tells me that tick diseases are killing off 90 per cent. of the cattle in some parts of the country: it is manifest, therefore, that these diseases will do an enormous amount of damage to colonising in that district. It is of great importance that we should know how the disease is spread, and, if possible, find some means to prevent it.

Some of the observations which Dr. Nuttall has made are of extreme importance and interest. One that struck me as very remarkable was that ticks kept for several months retain the parasite, and are able, on biting dogs, to produce disease. That shows how difficult it may be to stamp out the disease, if ticks retain their life so long.

With reference to Garrapato disease, I have recently had letters from a friend in Uganda, who has made some investigations as to that disease. He had six cases of it under observation; and after a very careful and prolonged examination, he was able to demonstrate the presence of a *spirillum* in all the cases. He has put forward the idea that this disease may be a *spirillum* one, and that on very good grounds: and it is by no means improbable that this tick disease is also spread by argas. My friend is going on with the work, and he will no doubt make further observations and experiments. The disease corresponds very closely to another disease, which comes after having slept in huts or houses in which ticks have been found. It is a fever, accompanied by intense pain and vomiting, and also by diarrhœa. After a few days the patient gets better, but there is often a relapse, with fresh attacks of fever. I think that is an exceedingly interesting observation, and one which will probably prove that the disease is really a *spirillum* disease.

Another point of interest one might mention is with respect to the question of blackwater fever, viz., the wonderful and peculiar local distribution of this disease in certain places. When I was in Uganda, there was a town at the end of the lake where everyone had blackwater fever. I have also known a special hill, and anyone living on this hill was almost certain to get blackwater fever, whilst the district all round was free from it. When I went there, there was no blackwater fever, but one day two cases occurred on this hill again. In the light of what we have heard, we may say that probably the ticks

always infect this hill: that they do not bite for some time, but that eventually they do bite and spread the disease.

There are plenty of ticks, of course, in Africa: it might even be this argas that has something to do with it, or some other tick. It is certainly a disease well worthy of further investigation, if one considers the vast mortality and loss of life due to this disease in Uganda and East Africa. It is spreading greatly, too, in Northern Nigeria.

There are many points in regard to the internal anatomy, and in regard to the development and spread of piroplasma through the tick, which still require working out. The anatomy is, as Dr. Nuttall has said, a very difficult subject, owing to the very strong integument which covers the insect. It is a subject that has apparently a large future before it, and we all look forward with great interest to future work on the tick question.

DR. NUTTALL: There is very little to say in response to what has been said. Sir Patrick Manson's suggestion with regard to blackwater fever is extremely interesting and very suggestive. It may be that we are dealing with parasites that are ultra microscopic—but it needs investigation.

With regard to Garrapato, I can only say that the question needs more investigation than has yet been given to it. I think that is where we stand at present with regard to all these things—they need more investigation. The subject of this disease is exceedingly suggestive, as also is this so-called disease due to argas, but we need more evidence—it is all very vague.

What Dr. Low has said brings out still more strongly the need of investigating these diseases. Dr. Philip Ross is now doing some work in this direction.

In answer to Professor Simpson, I may say that, unfortunately, quinine has no effect whatever, and at present nothing has been found that has any beneficial effect. A great many things have been tried, but nothing has been found to be of any use.

As to the remarks of Dr. Bentley, I would say that his description tallies exactly with what we have observed. Just as in cattle diseases, so in the tick, we have different forms of piroplasma.

A CONTRIBUTION TO THE ETIOLOGY OF RHEUMATIC FEVER.

By G. B. LONGSTAFF, M.D., F.R.C.P.

Dr. WHITELEGGE, C.B., President, in the Chair.

(*Read Friday, January 20th*, 1905.)

Nearly twenty-five years ago (7th April, 1880), I read a Paper before this Society entitled: "On some Statistical Indications of a Relationship between Scarlatina, Erysipelas, Puerperal Fever, and certain other Diseases."* Therein were published some of the results arrived at by comparing the curves expressing the fluctuations from year to year of the death-rates from a large number of assigned causes in England and Wales.

My conclusions were in that Paper rather hinted at than dogmatically expressed. Those to which I would now ask your attention were as follows:—(1) " . . . the curves of Erysipelas and Puerperal Fever are practically identical . . I confess that I find it difficult to avoid the conclusion that they are both due to one poison. Though the Puerperal Fever curve has great points of resemblance to the Scarlatina curve, it is far more like that of Erysipelas. It is also extremely like that of 'Rheumatism, with disease of heart or pericardium,' so that we may infer some close relationship to Rheumatic Fever, a point that I must reserve for further investigation, but may merely remark that the Erysipelas curve is still more like the Rheumatism curve.†

(2) " So far as I have been able to investigate the point, it does not appear that the heat of summer or the cold of winter varies either in a direct or inverse relation to the fatality of these diseases.‡ But when the amount of rain is examined from this point of view, it appears that there is some kind of inverse relationship between the two. The same result is obtained whether the amount of the fall, or the number of days on which it falls, is considered, the

* *Studies in Statistics.* London: Ed. Stanford, 1891, p. 310.

† *Ibid*, p. 316.

‡ "Fatality" here means "absolute number of deaths from," not "case mortality."

connection being somewhat closer in the latter case. . . . . This relationship is nearly as close as that which subsists between Diarrhœa and hot summers on the one hand, or Bronchitis and cold winters on the other,"* and it is strikingly suggestive.

This Paper was reprinted in my *Studies in Statistics*, together with an appendix, dated August, 1890, which last to some extent brought the figures up to date, but was mainly written to confute a paper by the late Dr. Matthews Duncan in the *Edinburgh Medical Journal* of March, 1876 (which I had not seen till after the reading of my first paper). Dr. Matthews Duncan sought to prove that Erysipelas and Puerperal Fever were not epidemic diseases. The source of Dr. Matthews Duncan's fallacy, as I considered it to be, was that, by a strange fatality, he *assumed* rheumatism to be a typical non-epidemic disease, and then compared its statistics with those of Erysipelas and Puerperal Fever; whereas I was led to *infer* that Rheumatism was probably an epidemic disease from the remarkable resemblance of the three.†

With a view to throwing further light upon the question, I constructed for my book a more complicated diagram, containing curves of the three diseases for each of the eleven registration divisions into which England and Wales are divided, and in commenting on it, said :‡ "If the diagram be looked at as a whole, or analysed in numerical detail, it will be found that the correspondences in nearly every case are far closer than mere coincidence would account for;" and added: "As regards Erysipelas and Puerperal Fever, it must, I think, be admitted that a relationship which holds good in the several parts of the country, as well as in the entire area, cannot be fortuitous."§

In 1902, Dr. Galabin, in his Presidential Address to the Hunterian Society,‖ applied himself to carrying on my methods for another twenty years, confirming my results in so remarkable a manner as to afford me much gratification. Unfortunately for me, Dr. Galabin has so completly taken the wind out of my sails that I feel much hesitation in troubling you with this paper to-night, and it is only the great importance of the subject that has led me to recur to it. Speaking of the curves showing the annual fluctuations of several diseases in England and Wales,

* *Studies in Statistics.* London: Ed. Stanford 1891, p. 322.
† *Ibid*, p. 324.
‡ *Ibid*, p. 330.
§ *Ibid*, p. 330.
‖ *Lancet*, 1902, p. 1,671.

Dr. Galabin said: "As regards the curve of Erysipelas, its most marked feature is its close resemblance, almost amounting to identity, to the curve of Puerperal Fever." Again he says: "A still more remarkable proof of the close relation of Erysipelas to Puerperal Fever will be found in comparing the curves of weekly variations (in London) . . . Not only is the mean curve for ten years practically identical for the two diseases, but the same is true of the very complicated curve showing the weekly variations in 1893 (the year of maximum mortality from Puerperal Fever). . . .* This identity becomes even more striking if the curves are compared with the totally different curve of Septicæmia and Pyæmia. This resemblance of the curves confirms the evidence of bacteriology as to the close connection between Puerperal Fever and Erysipelas. I think, indeed, that the inference may be carried somewhat further, and that it may be concluded that a variety of *Streptococcus*, closely allied to that of Erysipelas, is more generally concerned in fatal Puerperal Fever than in other forms of Septicæmia and Pyæmia, and that the contagion of Erysipelas is more dangerous to the puerperal woman than that of any other septic disease other than Puerperal Fever itself." Dr. Galabin adds some very suggestive reflections, *e.g.*: "There is a further question whether a year in which *Streptococci* are unusually abundant or easily diffused may not increase the mortality of Scarlet Fever by causing septic complications; and the same consideration would apply also to Rheumatic Fever." After commenting on the fact that during the last twenty years the curve of "Rheumatic Fever, Rheumatism of heart"—corresponds less closely with that of Erysipelas than under the old classification, in the earlier period, did that of "Rheumatism of the heart, or pericardium." Dr. Galabin goes on to say: "The diminution of similarity produced by the addition of Rheumatic Fever seems an argument in favour of the view that fatal heart affection in Acute Rheumatism is often due to *Streptococcic* complications, and that thus, Rheumatism of the heart, is more closely allied to septic affections than Rheumatic Fever in general."†

Dr. Richard Pritchard (*Public Health*, vol. 15, April, 1903, p. 388) attaches great importance to the influence of ventilation on the type of disease, and insists that the conditions which produce malignancy are identical with those which conduce to an attack; but he then, somewhat inconsistently, goes on to say: "It has been conclusively

* *Lancet*, p. 1,674. † *Ibid.*, p. 1, 675.

established by Dr. Marie Raskin and others, that the cause of those complications which give rise to malignant attacks of scarlet fever, diphtheria, etc—such as lymphadenitis, abscess, otitis, purulent synovitis, broncho-pneumonia, pleurisy, pyæmia, and septicæmia—is secondary infection of *Streptococcus pyogenes.* The same micro-organism is reputed to be the cause of hectic fever in phthisis."

If such secondary infection really occurs, it would explain many things that were once obscure, and amongst them the general resemblance between the curves of mortality from the diseases now under consideration.

I would here desire to call attention to the great change in medical opinion that has taken place since twenty-five years ago I ventured timidly to suggest that Rheumatic Fever was probably an epidemic disease.

The text-books in my hands as a student, putting out of consideration older authors, such as Sir Thomas Watson and Trousseau, were F. T. Roberts' *Handbook of Medicine,* 1873, and J. S. Bristowe's *Theory and Practice of Medicine,* 1876. Neither of those works, nor my voluminous notes of Charles Murchison's admirable Lectures, delivered in 1877, gave any hint that Rheumatic Fever should be etiologically classed with the other fevers. Indeed, Murchison went so far as to say: "The essential poison is not received from without." The causation, as then understood, was exposure to cold and wet, sudden chills, etc., while, at the same time, the presence of lactic acid in the blood was insisted on.

Now turn to the teaching of to-day. Professor W. Osler, in his *Principles and Practice of Medicine,** defines Rheumatic Fever as "an acute non-contagious fever, dependent upon an unknown infective agent," etc.; and without committing himself, reviews the evidence for regarding it as an acute infectious disease. Incidentally he quotes from Sir Wm. S. Church these words: "The curves of the mortality statistics . . . approximate very closely to those of pyæmia, puerperal fever, and erysipelas, diseases which are certainly associated with specific micro-organisms."† The last-named author, as I have unfortunately only recently learned, says: "The undoubted connection of Ulcerative Endocarditis—which, until recent years, was not recognised as distinct from simple rheumatic disease of the valves of the heart—with pyæmia and puerperal fever, throws some doubt on the correctness of the curves given by Longstaff,

* Fifth Edition, 1904, p. 166.
† *Lancet,* p. 168.

from statistics which began nearly forty years ago."* I should be the last to endeavour to minimise the importance of diagnosis, and the uncertainty introduced into medical statistics by carelessness or ignorance in the certification of the cause of death. However, the quite valid reproach of antiquity is removed from the figures that I shall lay before you to-night: figures which deal, for the most part, with Rheumatic Fever directly. Moreover, I have ascertained from Dr. J. Tatham that, until the beginning of the present century, deaths assigned to Malignant or Infective Endocarditis were classed under Valvular disease, and so altogether apart from Rheumatism. It may be remarked in this connection, that in the two years, 1901 and 1902, the deaths assigned to "Rheumatic Fever, Acute Rheumatism"—were more than three times as numerous as those assigned to "Rheumatism of Heart," and more than five times as numerous as those assigned to "Infective Endocarditis." Assuming similar proportions to have prevailed for the last twenty years, and that the deaths assigned to Rheumatism of Heart had been in reality *all* due to Ulcerative Endocarditis, they could only have influenced the resulting curve to the extent of 25 per cent. at the outside. But I doubt whether Sir Wm. S. Church would suggest that even a majority of these deaths were independent of Rheumatic Fever. Dr. J. Burney Yeo, in his *Manual of Medical Treatment*, 1903, says: "The disease known as Acute Rheumatism has been, by almost general consent, transferred to the class of acute infective diseases, and its probable microbic origin generally accepted."

The admirably exhaustive Milroy Lectures† of Dr. Arthur Newsholme must have had a not-inconsiderable share in bringing about this great change of opinion. Dr. Newsholme established the infective character of Rheumatic Fever by a mass of statistics derived from many countries, dealing, moreover, with prevalence as well as fatality. He was impressed by its association with dry seasons, and particularly with low ground-water. He termed it essentially a soil disease, and therein compared it with malaria: pointing out, however, that while Ague was usually associated with a moist soil, Rheumatic Fever was most prevalent when the subsoil was dry. Will the progress of our knowledge deal with Acute Rheumatism as drastically as it has done with Ague?

* Clifford Allbutt's *System of Medicine*, vol. iii, p. 3, 1897.

† *On the Natural History and Affinities of Rheumatic Fever: A Study in Epidemiology.*—"Lancet," March 9th, 1895, p. 589.

Lastly, Dr. F. J. Poynton, writing in Chalmers Watson's *Encyclopædia Medica*, 1902 (vol. x, p. 382), of the exciting cause of Acute Rheumatism, puts the case even more strongly when he says: "This in any case may be asserted with confidence, that the excitant is a bacterial infection . . . . essentially a *Diplococcus*."

Now this is very much more than a matter of definition. To attribute a disease mainly to chills, consequent on exposure to wet, or change of temperature, if it really be due to another cause or causes, may do harm in many ways. All are agreed that chills are hurtful, and that we should on general principles do well to avoid them. But if such a deadly disorder as Rheumatic Fever, with its army of victims suffering from chronic valvular disease of the heart, be attributed to the wrong cause, we may, while taking all the precautions dictated by the old etiology, be inadvertently exposing ourselves to the true cause. Moreover, therapeutics suffer, and remedies that are truly efficacious in mitigating the evil results of chills get far more credit than they deserve. This is an argument that obviously has an application far beyond the ailment under consideration.

Before proceeding to discuss the diagrams which I have constructed, it is necessary to clearly describe the changes in the method of dealing with the various assigned causes of death, grouped together under the vague heading *Rheumatism*, which were introduced by Dr. Ogle in 1881.

Under the old classification in vogue up to that time, the main heading *Rheumatism* included all deaths assigned to:

Rheumatic Fever.	Acute Rheumatism.
Rheumatism of Heart.	Sub-acute Rheumatism.
Rheumatic Arthritis.	Rheumatism unspecified.

This large group was in certain of the Tables subdivided by placing all deaths attributed to Rheumatic Fever, or to affections of the heart or pericardium directly associated with rheumatism (but not chronic valvular disease), into a sub-group named *Rheumatism of Heart or Pericardium*, the remainder forming the sub-group *Rheumatism*, which I shall hereafter, for the sake of clearness, speak of as *Residual Rheumatism*.

Under the new classification adopted in 1881, to the first group, now termed *Rheumatic Fever, Rheumatism of Heart*, were transferred *Acute Rheumatism* and *Sub-acute Rheumatism;* while *Residual Rheumatism* was still further diminished by the transfer of *Rheumatic Arthritis* to *Other*

Constitutional Diseases; to which also, on insufficient grounds as it appears to me, *Rheumatic Gout* was transferred from its old heading *Gout.*

This transference of *Acute Rheumatism* to its proper place as a synonym of *Rheumatic Fever* is not mentioned in the Table explaining the changes introduced in 1881, which forms an appendix to the report of that year; and it is due to the kindness of my friend, Mr. Noel A. Humphreys, of the General Register Office, that I am able to give this explanation. In a letter, dated April 20th, 1903, he said :— "Originally I believe the only cases noted to Rheumatism of Heart were those in which Fever or Heart Disease were certified in conjunction with Rheumatism, and even Acute Rheumatism was not so noted. I believe your difficulty (in accounting for the large increase subsequent to 1879) is due mainly to a change in the method of dealing with cases of Acute and Sub-acute Rheumatism, which were, until 1879 1880, ticked to Rheumatism and not noted to Rheumatic Fever, and possibly were not all classed to Rheumatic Fever until 1881."

TABLE I.—*England and Wales—Deaths.*

—	*Decennium*, 1871 to 1880. Old Classification.		—	*Decennium*, 1881 to 1890. New Classification.	
	Males.	Females.		Males.	Females.
Rheumatism ...	16,490	15,848	Rheumatism ...	17,310	17,523
Divided into : Rheumatism of Heart and Pericardium	7,283	6,965	Divided into : Rheumatic Fever (Acute Rheumatism), Rheumatism of Heart	13,063	12,492
Residual Rheumatism (including Acute Rheumatism)	9,207	8,883	Residual Rheumatism ...	4,247	5,031
Gout	4,370	1,337	Gout	4,350	1,119
—	—	—	Other constitutional diseases...	2,069	2,781

Numerically, the result of these changes was that, in 1881, and the years immediately following, while the deaths for *Rheumatism*, as a whole, continued to average about 3,400 annually, those attributed to *Rheumatic Fever, Rheumatism of Heart*—averaged just about 1,000 a year more than in the years immediately preceding 1880.

At the same time, the deaths from *Residual Rheumatism* fell by just about 1,000 a year, the transference of *Rheumatic Arthritis* to *Other Constitutional Diseases* producing no perceptible effect. This need excite no surprise when we learn that the last-named class covered only 379 deaths in 1881, a number that has since slowly and gradually increased to 637 in 1890, and further to 1,153 at the close of the century (see Table I). A method of analysis which I shall explain presently leads me to infer that the deaths classed under the vague heading, *Other Constitutional Diseases*, were, at any rate for the ten years 1881-1890, for the most part really due to *Gout* a conclusion which is to a great extent, confirmed by the fact that when Dr. Tatham, in 1901, created a new sub-class *Rheumatic Arthritis, Rheumatic Gout*—the number of deaths transferred thereto was 848, or 73 per cent. of the whole number contained in the previous year in the old class of *Other Constitutional Diseases.*

The year 1880 occupies an intermediate position, *Rheumatic Fever* gaining 500, while *Residual Rheumatism* lost 500, as compared with 1879. This quite agrees with Mr. Noel Humphreys's recollection that the transfer began the year before the new classification was put into general operation.

In what I have to say as to the figures for the last twenty years of the century that has just closed, it is, I fear, inevitable that I must repeat many things that Dr. Galabin has said; and I must ask your kind consideration and his forgiveness for so doing.

During this period of twenty years—1881-1900 inclusive—the total number of deaths in England and Wales attributed to "Rheumatic Fever, Rheumatism of the Heart"—was 51,666.

Of these deaths, 26,085 were of males, and 25,089 of females, or for every 1,000 males that died, there were 981 females.

This, however, gives quite a wrong impression, for if the number of each sex living be taken into consideration, it will be found that the average male death-rate was 92 per million, while the average female death-rate was only 84.

But these 51,666 deaths by no means afford a complete measure of the evil wrought by Rheumatic Fever in twenty years, for during the same period 171,298 persons died whose deaths were certified as due to Valvular Disease of the Heart; and an unknown but certainly a very large proportion of these persons might rightly have attributed their long drawn-out sufferings, mental as well as bodily, to an antecedent attack of Rheumatic Fever.

The *Geographical Distribution* of these deaths is given roughly in Table II.

TABLE II.—*Death-Rates per Million Persons living in Registration Divisions.*

1881 to 1890.		1891 to 1900.	
North-Western ...	109	North-Western ...	101
London	101	Wales	91
Yorkshire	98	London	89
South-Eastern ...	89	Yorkshire	86
West Midland... .	88	West Midland, Northern ...	81
South Midland, South Western ...	87	South Midland ...	79
Wales	86	South-Eastern... ...	77
Northern	83	South-Western ...	76
Eastern, North Midland ...	81	Eastern, North Midland ..	74
ENGLAND AND WALES ...	93	ENGLAND AND WALES ...	85

This gives the crude Death Rates for the Registration Divisions, calculated on the mean populations between two successive censuses. It will be noted that Rheumatic Fever was in each decade most fatal in the North-Western Division, and least fatal in the Eastern and North Midland Divisions. London and Yorkshire were near the top of the list in each period. The main difference between the two decades was that the death-rate was notably less in the second, save in Wales. In conformity with this, the chief change in the order is that the Welsh division sprang up from below the middle to the second place.

I do not see anything in the geographical distribution of the disease to support the "Chill" theory of its etiology, and greatly suspect that statements in the older books as to predisposing localities and occupations owed more to the theory than to precise investigations. The prevalence of the disease would appear to depend more upon density of population than relative humidity.

There is a way of examining the Registrar-General's statistics of the causes of death which admits of graphic representation and gives interesting results, though it is seldom employed.

In Diagram A are represented the average number of deaths in England and Wales from certain assigned causes at various groups of Ages, for the two consecutive decennia 1871-80 and 1881-90, males and females being given separately. The periods chosen are the last decennium of the old classification and the first of the new. (See Tables III and IV.)

In order to understand clearly the method of representa-

tion adopted, it should be explained that the orange-coloured area in the top left-hand corner deals with the 16,490 deaths of males from the comprehensive alleged cause *Rheumatism*, as used in the shorter, or more generalised classification, of the earlier period. The whole area is divided into seventeen vertical columns, representing eleven age-periods, one column being assigned to each of the five earlier periods of five years each, and two columns to each of the five periods of ten years each, and two columns to all the deaths occurring at the age of 75 and upwards. In every case each small oblong space corresponds to an average of 10 deaths per annum.

Immediately below is a similar diagram dealing with the 15,848 deaths of females. Neither diagram indicates any clear law of incidence, but markedly more females than males die in youth, while the disease is less fatal in middle life to women than to men.

To the right are shown similar diagrams for the later period, dealing with 17,310 deaths of males and 17,523 deaths of females respectively. The diagrams for the two periods correspond closely, but the deaths of males at the age group 10-15 are both relatively and absolutely more numerous in the second period.

Now turn to the crimson diagrams on the left-hand side, representing the deaths of 7,283 males, and 6,965 females assigned to *Rheumatism of the Heart and Pericardium*, and, as explained above, including deaths attributed to *Rheumatic Fever* but *not* deaths attributed to *Acute*, or *Sub-acute, Rheumatism.*

The two diagrams are very similar, and we now see clear indications of a definite law of incidence. The disease kills most persons, in both sexes, in youth, between the ages of 10 and 20, and is less and less fatal as age advances.

Next look at the yellow diagrams on the left-hand side. These represent the deaths from *Residual Rheumatism*, 9,207 males and 8,883 females, including, be it remembered, those attributed in the death certificates to *Acute*, or *Sub-acute Rheumatism.* The construction of the diagrams is such that the sum of corresponding crimson and yellow ordinates is equal to the orange ordinate of the upper diagram. It will be observed that these yellow diagrams differ from the corresponding orange diagrams, mainly in the fact that the peak or hummock corresponding to deaths in youth has disappeared; indeed, two-thirds of the deaths at ages 5 to 20 appear in the red and only one-third in the

yellow diagram. Conversely, at ages 45 to 65 two-thirds of the total deaths appear in the yellow diagram, and at more advanced ages, even $\frac{4}{5}$ and $\frac{7}{8}$. In other words, Rheumatism of the Heart (including Rheumatic Fever) is a disease that has its greatest incidence on youth, while Residual Rheumatism is most fatal to the old, especially old women, but has a comparatively uniform incidence from youth throughout middle life.

Let us now turn to the crimson and yellow diagrams on the right-hand side, dealing with deaths of the second period, arranged in accordance with the new classification. The essential point to bear in mind is that deaths certified to Acute or Sub-acute Rheumatism are now classed with those assigned to Rheumatic Fever, or Rheumatism of the Heart. What do we see as a consequence? While the diagram of Rheumatic Fever gives again a fairly good curve* (quite a good curve in the case of females), culminating as before in youth, the diagram of Residual Rheumatism gives an even more regular curve, culminating in the age-period, 65 to 75. It seems impossible to imagine any clearer proof that Rheumatic Fever and Residual Rheumatism are truly distinct causes of death, and that the transfer was more than justified.

For comparison I have added diagrams representing the deaths classed to Gout and (in the case of the second period) to Other Constitutional Diseases, *i.e.*, Rheumatic Gout and Rheumatoid Arthritis. The exigencies of space have compelled me to put the female curve on the left-hand side. The term "Rheumatic Gout" is, I feel sure, in the vast majority of cases applied to cases of Gout pure and simple, and the figures suggest that a very large proportion of the deaths coming under the heading that I term Residual Rheumatism are also essentially gouty in their nature. Rheumatoid Arthritis is, of course, an entirely distinct disease, having a distinct pathology, but every person that I have ever come across stating that his medical man had told him that he had Rheumatic Gout was, in my judgment, suffering from Gout, pure and simple. It appears to me that cases of Gout in which deformity of the joints is a more obvious symptom (especially in women) than pain, are apt to be labelled with that most objectionable—because unscientific—name, Rheumatic Gout.

Diagram B is somewhat complicated, and needs careful

* Good in the mathematical sense of the term "curve," the graphic expression of a numerical law, as distinguished from the irregular so-called "curve," expressing, *e.g.*, the irregular variations from year to year.

examination. The curves apply to England and Wales, and represent the fluctuations from year to year in the Death-Rates per million Persons living, from a number of assigned causes. (See Tables VII, VIII, IX, X, and XII.)

I will ask you, in the first place, to confine your attention to the portion on the right hand of the dividing line, which deals with the period of twenty years—1881-1900.

As in all my diagrams, the mean rates for the period are given in figures at the side, and shown by dotted lines. The curves represent deviations from the mean, so that each small division of the vertical scale corresponds to 10 per cent. above or below. In this way all the curves, whether relating to small or large numbers, are strictly comparable.

To the left of the dividing line are given the curves, taken from my former paper,* relating to the twenty-five years—1856-1880, inclusive. I have omitted the year 1855, chiefly from considerations of space, and would premise that these curves are not quite accurately represented, since the mean from which the percentage deviations are calcalculated is that of the twenty-four years—1855-1878. The only difference caused by this discrepancy is that some of the curves are placed a trifle higher, or lower, in reference to the dotted lines, than they should be, but the argument is in no way affected.

In the case of Enteric Fever, the deviations have been calculated from the mean of thirty-two years—1869-1900, and in the case of Diphtheria I have, for the earlier period as for the later, included Cynanche Maligna, thereby slightly modifying the earlier years of my old diagram, and the entire curve is now calculated from one mean, that, namely, of the whole forty-five years. The continuity of these curves is indicated by the breaks in the dividing black line.

All the other curves for the earlier period are calculated from the means given on the left edge of the diagram, taken from my *Studies in Statistics*. Hence it follows that (with the exceptions of Enteric Fever and Diphtheria) the curves on either side, though of similar colours, must not be regarded as continuous, for in some cases the classification differs, (notably so in the case of Rheumatism), and in all cases the means, from which their percentage fluctuations are calculated, differ. To indicate this, and to make some sort of comparison possible, I have, down the centre of the diagram, given the actual death-rates per million for the last year of the earlier period, and the first year of the later period.

* As reprinted in *Studies in Statistics*.

Reverting to the right-hand side of the diagram, certain features strike the eye at once. First, all the curves (with the single marked exception of that of Diphtheria) exhibit a downward tendency, most conspicuous in the cases of Scarlet Fever and Erysipelas. Secondly, all the curves, with the notable exception of Residual Rheumatism show an outstanding peak corresponding to the year 1893. This in the cases of Puerperal Fever and Diphtheria, was the year of maximum mortality, but in the case of the other diseases, only a more or less well-marked secondary maximum. The dry and hot summer of 1893 led also to a very high death-rate from Epidemic Diarrhœa, but the annual fluctuations in the curve of that disease have usually but little correspondence with those that we are now treating of. Moreover, its fluctuations from year to year are in comparison extreme.

If we now turn to the left-hand side of the diagram, which deals with the twenty-five years, 1856-1880, we see that the fall in the curves of Scarlatina and Erysipelas during the last twenty years is a continuation of that which commenced in the later 'seventies; in fact, the tale of deaths has almost continuously decreased since the fatal year 1874.

In the case of Puerperal Fever, we find a different state of things. The mean for the later period—76 deaths per million—is just 20 per cent. higher than that for the earlier series of years, when it was but 63. Although it is true that the death-rate, even in 1893 (when it was 102 per million) was below that of 1875 (105 per million), and considerably below that of 1874 (when it reached 131), still Puerperal Fever was, in the aggregate, far more fatal in the eight years, 1881-1888, than at any previous time of like duration for which figures are available, and even during the last six years has scarcely fallen to the rates prevailing prior to the great epidemic of 1874. The letters of enquiry instituted by the Registrar-General in 1881, are responsible for an increase of somewhere about 10 per cent.,* but that will only in part account for the greater fatality.

The curve of Scarlatina exhibits curious fluctuations of a secular character. During the period covered by my former paper, the curve rose and fell in great waves, showing five hollows, from 30 to 50 per cent. below the mean of 854, and four crests of from 23 to 70 per cent. above it. The curve then fell each year—1881, 1882,

* *Studies in Statistics*, p. 325.

1883, 1884, and 1885—and since then has fluctuated, though in a far less majestic manner than before, about a mean rate of 183 (for the eighteen years, 1885-1902). How this astonishing fall of the mean to less than a fourth of its former magnitude is to be explained, how much of it is to be attributed to segregation in hospitals, how much to improved treatment, how much to change of type, I do not pretend to know, but am inclined to give much credit to the last. A very similar, and scarcely less remarkable, secular change is shown by the curve of Enteric Fever (not treated of in my previous paper). The figures are only available from 1869 onwards. For the first seven years the death-rate was almost steady, averaging 378 per million; then for nine years it fell irregularly, but for the eighteen years—1885-1902—it has exhibited moderate fluctuations (about 25 per cent. above or below) from a mean of 175, less than half that of the early 'seventies. It will be observed that the secular changes since 1875 in the cases of Scarlatina and Enteric Fever though differing greatly in degree, show steady diminution and comparative stability during identical periods.

Owing to the changes in the classification detailed above, no useful comparison can be made between the curves of Rheumatic Fever for the two periods.

If the curves in Diagram B be carefully compared, each with each, it will be found that those of Erysipelas and Puerperal Fever are so alike as to be practically identical, for the greater part of their course at any rate, and this was even more the case in the previous period. The curve of Rheumatic Fever is very like both these, but follows that of Puerperal Fever somewhat the more closely. On the contrary, Scarlatina, while it has a strong resemblance to all three, is most suggestive of Erysipelas.

The curve of Septicæmia, which includes all non-puerperal cases assigned to Pyæmia, is in some respects more like that of Scarlatina than that of Erysipelas. This curve has been greatly raised during the last twenty years by the change of practice, whereby deaths from Pyæmia following accidents, which were formerly included under *Violent Deaths*, are now classed to the resulting intercurrent malady. The increase amounts to an average of about 50 per cent.

The pale-yellow curve at the bottom of Diagram B. shows the fluctuations of Residual Rheumatism for the two periods. That to the left hand shows that in the earlier period this was like a flattened or foreshortened shadow of the curve of Rheumatism of the Heart with the notable

exception that the latter rises in the last year whereas the former falls. The corresponding curve on the right-hand side, that for the later period, oscillates about a mean, that is, roughly speaking, one-half that of the first period (: 32 : 70), but these oscillations *bear no relation whatever to those of Rheumatic Fever.* The lowering of the mean is of course due to the transference of deaths assigned to Acute or Sub-acute Rheumatism to the heading Rheumatic Fever. Moreover, the discrepancy alluded to above in the last year of the first period is due to this change in classification having commenced in the year in question. (See Tables V and VI.)

Now let us turn to Diagram C. This deals with seasonal variations, and exhibits the mean Weekly Deaths in London for almost the same period —1882-1901—drawn in like manner, so that the ordinates express a deviation above or below the weekly mean, each division corresponding to 10 per cent. It should, however, be explained that, with a view to removing fortuitous irregularities owing to the small numbers dealt with, the curve has been *smoothed* by the process of taking three continuous means; that is to say, the figure set down for each week is the mean of that for the week in question, that for the preceding, and that for the following week.

It will be observed that the three curves for Erysipelas, Puerperal Fever and Rheumatic Fever very closely resemble one another: this was also the case in the earlier period. That of Rheumatic Fever approaches more closely to Erysipelas than to Puerperal Fever; the former occupying an intermediate position. All the curves are well above the average throughout the three months, November, December, and January, and all are well below the average throughout May, June, July and August. The weeks in which the actual maxima and minima are found differ somewhat, it is true, but the general resemblance is nevertheless sufficiently striking.

The corresponding Diagram (Plate XX) in my *Studies in Statistics* gave very similar results, but the improvements in classification introduced in 1881 have somewhat increased the resemblance of the Rheumatic Fever curves to the others.

At the bottom of Diagram C are given, in grey and black respectively, the curves for Residual Rheumatism and Gout.* It will be noted that, while generally resembling

* As the figures are very small, it was necessary in order to *smooth* them adequately to take *five* continuous means.

one another, they differ essentially from the other curves. Unfortunately, the "Other Constitutional Diseases" of the Weekly Returns include a number of diseases having nothing in common, and so differ from the groups bearing the same name in the Annual Reports. (See Tables XI and XII.)

Reverting to Diagram B, it will be seen that at the top and bottom two inverted curves are constructed by drawing blue ordinates downwards. These represent the deviation from the mean of the Greenwich Rainfall. As in my former Paper, to prevent exceptional storms having an undue influence upon the curve, I have in its construction combined the number of inches measured with the number of days on which rain was measured, adding together half the percentage of each.

No close relationship is observed, such as was seen in the previous twenty years, but it will be noted that the fatal year, 1893, was dry, and that it was followed by a very wet year. (See Tables XI and XIII.)

I then divided England and Wales into three regions—

(1) South Eastern, comprising the London, South Eastern, South Midland, and Eastern registration divisions, with an aggregate mean population of some 10½ millions.

(2) Western and Midland, comprising the South Western, West Midland, North Midland, and Welsh registration divisions, with an aggregate mean population of 8¾ millions.

(3) Northern Region, comprising the North Western, Yorkshire, and Northern registration divisions, with a mean aggregate population of nearly 9¾ millions.

I constructed a Rheumatic Fever curve for each of these regions, and with them compared Rain curves for Greenwich, Wolverhampton, and Leeds respectively.

These last three differed considerably one from the other, but the three Rheumatic Fever curves were much more concordant than might have been expected.

Curiously enough, the Rheumatic Fever curve for the South Eastern region did not show the inverse connection with the Greenwich Rainfall more closely than the curve for England and Wales. (See Table XII.)

I made yet a third set of comparisons by taking out the Greenwich Rainfall for the first four months of the year, the second four months, and the third four months. In this case I found that the closest connection with the Rheumatic Fever curve was with the Rain curve for the four months, May, June, July and August. This curve is

given at the bottom of the diagram. The inverse connection is not very close. It may be asserted that epidemics of Rheumatic Fever occur in, or follow, dry summers, but every dry summer does not produce an epidemic. Some condition was wanting in the very dry summer of 1899, which prevented more than a trifling rise in the diseases here treated of.

But when stating that the relations between the diseases we are here treating of and rainfall were less marked in the later than in the former period, it should be noted that the fluctuations in the rainfall itself have been far less remarkable in recent years. A cause that is at the most secondary could not be expected to produce strictly proportional variations; it might well be that only extreme fluctuations of the rainfall would materially affect the prevalence or fatality of Rheumatic Fever or Scarlatina.

I think that I shall be justified in drawing the following inferences from the Registrar-General's figures:

(1) An analysis of the ages at death proves that alterations in the classification of causes of death, introduced about 1881, whereby deaths certified as due to "acute rheumatism," and "sub-acute rheumatism," were grouped with those assigned to "rheumatic fever" and "rheumatism of the heart or pericardium," bring the data more into accord with etiological facts than before.

(2) The same analysis gives reason to believe that at least a large majority of the deaths certified as due to "rheumatic gout" were really due to "gout."

(3) The geographical distribution of rheumatic fever gives no support to the old idea that it is mainly caused by cold, wet, or chills. It would appear to depend rather upon density of population.

(4) The curves expressing the year-to-year mortality from scarlatina, erysipelas, puerperal fever, septicæmia, rheumatic fever, and enteric fever all show a downward tendency.

(5) In both periods, viz., 1856-1880, and 1881-1900, the curves of erysipelas and puerperal fever are so alike as to be practically almost identical.

(6) In the second period, the curve of rheumatic fever closely resembles the curves of erysipelas and puerperal fever.

(7) The curves of scarlet fever and enteric fever exhibit notable secular variations. The former for the years 1856-1880 fluctuated widely about a mean of 850 deaths per million. It then fell steadily till 1885, since which time it has fluctuated slightly about a mean of 183.

(8) The enteric fever rate was nearly constant for the seven years 1869-1875, averaging 378 per million. It then fell for nine years irregularly, but for the eighteen years, 1885-1902, it exhibited moderate fluctuations, about a mean of 175 per million.

(9) Rheumatic fever, and the diseases here compared therewith, show a rough inverse relationship to rainfall, but this is not so marked in the latter period as in the earlier. A dry summer appears to favour an epidemic of rheumatic fever, though it is not invariably followed by one.

(10) In London, rheumatic fever, erysipelas, and puerperal fever have almost identical seasonal distribution; and this holds equally for an average of ten years, and for the epidemic year 1893.

In conclusion, no one is so conscious as myself that a good deal of labour has produced but very small results; yet since these results are founded on a very wide basis of facts, they deserve the most careful consideration of the etiologist. The very great importance of rheumatic fever, not merely as itself the direct cause of much suffering, but from the lasting misery due to its characteristic complication, endocarditis, must be my excuse for taking up so much of your time.

TABLE III.—ENGLAND AND WALES.

Average Annual Number of Deaths at Certain Ages.
Decennium 1871-80.
[Compare Diagram A.]

Age.	Rheumatism.		Rheumatism of Heart, etc.		Residual Rheumatism.		Gout.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
0 –	20	18	10	10	10	9	—	—
5 –	105	92	70	61	35	30	0.1	—
10 –	129	150	88	103	41	47	—	—
15 –	139	147	93	99	46	48	0.1	—
20 –	119	121	67	75	51	46	0.5	—
25 –	243	187	123	98	121	89	7	3
35 –	234	169	102	77	132	92	31	7
45 –	183	171	68	67	116	108	80	18
55 –	193	210	55	54	138	155	126	37
65 –	192	216	41	40	150	176	136	46
75 –	92	104	11	12	81	93	57	23
Total ...	1649	1585	728	696	921	888	437	134

TABLE IV.—ENGLAND AND WALES.

Average Annnal Number of Deaths at Certain Ages.
Decennium 1881-90.
[Compare Diagram A.]

Age.	Rheumatism.		Rheumatic Fever, etc.		Residual Rheumatism.		Gout.		Other Constitutional Diseases.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
0 –	29	26	27	25	2	2	...	...	2	2
5 –	114	111	111	109	3	2	...	...	1	1
10 –	152	164	148	159	3	4	...	...	1	1
15 –	154	160	148	156	6	5	...	...	2	2
20 –	119	132	112	126	6	6	0.1	...	2	3
25 –	240	210	222	194	18	16	5	2	7	6
35 –	246	188	212	154	35	34	27	4	16	17
45 –	186	193	137	134	49	58	65	12	31	42
55 –	193	217	99	98	94	119	118	25	50	78
65 –	198	237	67	71	131	166	143	41	66	85
75 –	100	113	23	23	78	91	77	28	31	42
Total	1731	1752	1306	1249	425	503	435	112	207	278

TABLE V.—LONDON. AVERAGE WEEKLY DEATHS (PERSONS).

Twenty Years—1882-1901. [Compare Diagram C.]

Weeks.	Rheumatic Fever, etc.			Erysipelas.			Puerperal Fever.			Residual Rheumatism.			Gout.		
	Average Deaths.	Per Cent. of Mean.	Smoothed Figures.*	Average Deaths.	Per Cent. of Mean.	Smoothed Figures.*	Average Deaths.	Per Cent. of Mean.	Smoothed Figures.*	Average Deaths.	Per Cent. of Mean.	Smoothed Figures.†	Average Deaths.	Smoothed Figures.†	Per Cent. of Mean.
1st	8.20	111	115	5.80	119	128	6.55	135	131	3.25	152	109	3.30	3.35	116
2nd	8.50	115	119	7.05	144	128	7.25	149	148	2.20	102	113	3.65	3.55	123
3rd	9.25	125	117	5.80	119	122	5.55	161	148	2.15	100	118	3.55	3.70	128
4th	8.30	112	117	5.00	102	105	6.55	135	134	2.55	119	108	3.85	3.59	124
5th	8.30	112	113	4.55	93	105	5.20	107	121	2.55	119	110	4.15	3.58	124
6th	8.50	115	109	5.95	122	102	5.85	120	110	2.10	98	118	2.75	3.60	125
7th	7.30	99	107	4.40	90	96	5.05	104	108	2.45	114	116	3.60	3.42	118
8th	7.95	108	101	3.80	77	90	4.80	99	102	3.05	142	119	3.65	3.13	108
9th	7.10	96	100	4.95	101	84	5.10	105	101	2.35	109	125	2.95	3.23	112
10th	7.10	96	95	3.60	74	89	4.85	100	99	2.85	132	125	2.70	3.21	111
11th	6.95	94	94	4.55	93	83	4.55	94	88	2.70	126	117	3.25	3.16	109
12th	6.70	91	96	4.05	83	86	3.40	70	89	2.45	114	112	3.50	3.15	109
13th	7.70	104	92	4.05	83	89	5.10	105	93	2.20	102	112	3.40	3.21	111
14th	6.05	82	92	5.00	102	94	5.00	103	100	1.80	84	105	2.90	3.21	111
15th	6.70	91	86	4.75	97	92	4.45	92	97	2.90	135	112	3.00	3.17	110
16th	6.40	87	89	3.75	77	82	4.70	97	95	1.95	91	111	3.25	3.12	108
17th	6.65	90	80	3.55	73	80	4.65	96	97	3.15	146	118	3.30	3.07	106
18th	4.60	62	82	4.40	90	80	4.75	98	95	2.15	100	111	3.15	3.02	104
19th	7.00	95	83	3.80	78	88	4.50	93	95	2.55	119	110	2.65	3.02	104
20th	6.85	93	87	4.75	97	80	4.65	96	92	2.10	98	99	2.75	3.09	107
21st	5.40	73	84	3.15	64	80	4.20	86	87	1.85	86	99	3.25	3.04	105
22nd	6.35	86	78	3.80	78	74	3.90	80	90	1.95	91	89	3.65	3.18	110

23rd	5.55	75	88	3.95	81	79	5.05	104	83	2.15	100	83	2.90	3.12	108
24th	7.50	101	88	3.90	80	82	3.20	66	85	1.55	72	79	3.35	3.03	105
25th	6.40	87	88	4.20	86	77	4.20	84	74	1.40	65	78	2.45	2.70	93
26th	5.60	76	81	3.25	67	71	3.55	73	81	1.45	67	78	2.80	2.71	94
27th	6.05	82	82	3.00	61	69	4.15	85	77	1.85	86	79	2.00	2.54	88
28th	6.55	89	85	3.85	79	69	3.55	73	79	2.15	100	82	2.95	2.66	92
29th	6.25	85	83	3.25	67	73	3.85	79	77	1.65	77	84	2.50	2.47	85
30th	5.65	76	77	3.55	73	78	3.85	79	79	1.70	79	86	3.05	2.61	90
31st	5.25	71	77	4.65	95	83	3.75	77	77	1.70	79	89	1.85	2.44	84
32nd	6.10	83	85	3.95	81	88	3.60	74	85	2.05	95	91	2.70	2.53	88
33rd	7.50	101	94	4.30	88	90	5.10	105	87	2.45	114	89	2.10	2.39	83
34th	7.25	98	96	4.90	100	90	4.00	82	93	1.35	86	95	2.95	2.48	86
35th	6.60	89	91	3.95	81	89	4.45	92	83	1.55	72	89	2.35	2.39	83
36th	6.40	87	91	4.15	85	88	3.60	74	79	2.35	109	85	2.30	2.41	83
37th	7.15	97	95	4.75	97	97	3.45	71	77	1.40	65	83	2.25	2.28	79
38th	7.50	101	99	5.30	108	101	4.15	85	80	2.05	95	89	2.20	2.25	78
39th	7.25	98	102	4.75	97	105	4.05	83	88	1.60	74	84	2.30	2.31	80
40th	7.85	106	102	5.30	108	104	4.65	96	95	2.15	100	92	2.20	2.29	79
41st	7.45	101	113	5.25	107	119	5.15	106	104	1.85	86	90	2.60	2.40	83
42nd	9.70	131	119	6.85	140	129	5.30	110	112	2.30	107	90	2.15	2.49	86
43rd	9.30	126	131	6.85	140	140	5.80	119	125	1.80	84	93	2.75	2.55	88
44th	10.05	136	135	6.85	140	142	7.15	146	129	1.55	72	95	2.75	2.51	87
45th	10.55	143	133	7.15	146	141	5.90	121	129	2.50	116	94	2.50	2.56	89
46th	9.00	122	129	6.65	136	134	5.80	119	125	2.10	98	93	2.40	2.72	94
47th	9.05	122	120	5.85	120	127	6.55	135	124	2.20	102	105	2.40	2.72	94
48th	8.45	114	116	6.15	126	131	5.70	117	124	1.65	77	106	3.55	2.78	96
49th	8.20	111	117	7.20	147	141	5.90	121	119	2.80	130	106	2.75	2.87	99
50th	9.30	126	122	7.30	149	146	5.80	119	120	2.65	123	105	2.80	3.07	106
51st	9.65	131	123	6.90	141	137	5.85	120	116	2.15	100	120	2.85	3.02	104
52nd	7.60	103	120	5.75	118	127	5.25	108	121	2.00	93	114	3.40	3.20	111
Mean	**7.40**	...	...	**4.89**	...	...	**4.86**	...	...	**2.14**	...	...	**2.89**	—	—

* Each figure the mean for that, the preceding, and the following weeks.

† Each figure the mean of that, the two preceding, and the two following. In the case of Gout the Weekly Deaths were Smoothed instead of the percentages.

TABLE VI.—LONDON: THE YEAR 1893.

[Compare Diagram C.]

Week.	Rheumatic Fever, etc.			Erysipelas.			Puerperal Fever.		
	Deaths each Week.	Smoothed Figures.	Per Cent. of Mean.	Deaths each Week.	Smoothed Figures.	Per Cent. of Mean.	Deaths each Week.	Smoothed Figures.	Per Cent. of Mean.
1st	10	7.00	94	10	10.00	204	10	8.00	164
2nd	9	9.00	121	11	9.67	198	8	8.00	164
3rd	8	8.00	108	8	7.00	143	6	6.67	137
4th	7	8.33	112	2	5.33	109	6	6.00	123
5th	10	8.67	117	6	6.00	123	6	7.33	151
6th	9	10.00	134	10	7.00	143	10	7.33	151
7th	11	8.00	108	5	6.00	123	6	6.33	130
8th	4	6.33	85	3	4.33	89	3	5.00	103
9th	4	5.00	67	5	3.67	75	6	5.33	110
10th	7	6.00	81	3	3.33	68	7	6.33	130
11th	7	8.33	112	2	3.00	61	6	6.00	123
12th	11	8.00	108	4	3.67	75	5	6.00	123
13th	6	7.00	94	5	3.67	75	7	5.00	103
14th	4	5.33	72	2	4.33	89	3	4.67	96
15th	6	5.33	72	6	3.67	75	4	4.00	82
16th	6	6.67	90	3	3.33	68	5	4.33	89
17th	8	7.00	94	1	2.67	55	4	4.33	89
18th	7	8.00	108	4	3.67	75	4	4.67	96
19th	9	6.67	90	6	4.67	96	6	5.33	110
20th	4	7.00	94	4	4.33	89	6	6.00	123
21st	8	6.00	81	3	4.00	82	6	4.67	96
22nd	6	6.67	90	5	4.33	89	2	6.00	123
23rd	6	7.67	103	5	5.67	116	10	5.00	103
24th	11	9.00	121	7	9.00	184	3	6.00	123
25th	10	8.33	112	15	8.00	164	5	3.00	62
26th	4	8.00	108	2	7.67	157	1	6.00	123
27th	10	8.67	117	6	4.00	61	12	5.33	110
28th	12	10.33	139	4	5.00	102	3	6.00	123
29th	9	10.33	139	5	6.00	123	3	3.00	62
30th	10	10.00	134	9	7.67	157	3	3.67	75
31st	11	11.67	157	9	9.00	184	5	5.33	110
32nd	14	11.67	157	9	11.00	225	8	8.33	171
33rd	10	14.67	197	15	11.00	225	12	9.00	185
34th	20	13.33	179	9	11.00	225	7	7.67	158
35th	10	13.33	179	9	9.33	191	4	5.33	110
36th	10	8.67	117	10	8.67	177	5	4.33	89
37th	6	11.33	152	7	8.00	164	4	5.67	117
38th	18	11.33	152	7	7.33	150	8	5.67	117
39th	10	12.33	166	8	9.00	184	5	6.33	130
40th	9	10.67	143	12	9.67	198	6	8.00	164
41st	13	11.67	157	9	9.00	184	13	9.67	199
42nd	13	13.00	175	6	10.33	211	10	12.33	254
43rd	13	14.67	197	16	13.33	273	14	13.33	274
44th	18	16.67	224	18	16.33	334	16	12.00	247
45th	19	17.00	228	15	14.33	293	6	11.67	240
46th	14	15.33	206	10	13.67	280	13	9.33	192
47th	13	14.00	188	16	13.33	273	9	9.00	185
48th	15	12.33	166	14	14.67	300	5	8.67	178
49th	9	13.00	175	14	13.33	273	12	9.00	185
50th	15	10.33	139	12	18.33	375	10	8.67	178
51st	7	13.00	175	29	16.67	341	4	8.00	164
52nd	17	10.00	134	9	16.00	327	10	7.00	144
Mean of 20 years	...	7.44	...	...	4.89	...	...	4.86	—

TABLE VII.—ENGLAND AND WALES.

Deaths and Death-Rates, 25 *Years*, 1856-1880.

[Compare Diagram B.]

Year.	Scarlatina.			Septicæmia, etc.			Erysipelas.		
	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.
1856	13,463	707	83	...	...	...	1998	105	115
1857	12,558	652	76	...	...	...	1478	77	84
1858	23,600	1212	142	...	...	...	2000	103	112
1859	19,221	976	114	...	...	...	1924	98	107
1860	9,681	486	57	...	...	...	1662	84	92
1861	9,077	451	53	...	...	...	1539	76	83
1862	14,834	728	85	69	3	21	1520	75	82
1863	30,475	1478	173	159	8	47	1906	92	100
1864	29,700	1422	167	202	10	59	2091	100	109
1865	17,700	837	98	242	11	70	1953	92	100
1866	11,685	546	64	273	13	78	1665	78	85
1867	12,300	567	66	246	11	70	1445	67	73
1868	21,912	998	117	337	15	94	1942	88	96
1869	27,641	1244	146	355	16	98	1853	83	91
1870	32,543	1446	169	407	18	111	2120	94	103
1871	18,567	815	96	375	16	101	2216	97	106
1872	11,922	517	61	366	16	98	1771	77	84
1873	13,144	563	66	405	17	107	2027	87	95
1874	24,922	1054	123	674	29	175	3358	142	155
1875	20,469	855	100	645	27	166	3017	126	138
1876	16,893	697	82	549	23	139	2327	96	105
1877	14,456	589	69	584	24	146	2077	85	93
1878	18,842	758	89	577	23	143	1870	75	82
1879	17,613	694	81	643	25	156	1824	72	78
1880	17,404	675	79	645	25	154	2014	78	85
Mean of 24 years, 1855-78	18,435	854	100	380*	16	100	1994	92	100

* Mean of 17 years 1862-78.

TABLE VII.—ENGLAND AND WALES (*Continued*).

Deaths and Death-Rates, 25 *Years*, 1856-1880.

[Compare Diagram B.]

Year.	Puerperal Fever.			Rheumatism of Heart, etc.			Residual Rheumatism.		
	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.
1856	1067	56	89	681	36	78	1326	72	102
1857	836	43	69	498	26	56	1190	63	89
1858	1068	55	88	667	34	74	1275	67	95
1859	1238	63	101	784	40	86	1340	69	98
1860	987	50	80	753	38	82	1245	64	91
1861	886	44	70	661	33	71	1321	66	94
1862	940	46	73	704	35	75	1239	60	85
1863	1155	56	89	870	42	91	1305	63	89
1864	1484	71	113	1001	48	104	1558	75	106
1865	1333	63	101	951	45	98	1579	75	106
1866	1197	56	89	846	40	86	1492	69	98
1867	1066	49	78	793	37	79	1463	67	95
1868	1196	54	86	1071	49	106	1432	65	92
1869	1181	53	85	1160	52	113	1593	72	102
1870	1492	66	105	1230	55	119	1561	69	98
1871	1464	64	102	1203	53	114	1523	67	95
1872	1400	61	97	1049	45	99	1407	61	87
1873	1740	74	118	1034	44	96	1469	63	89
1874	3108	131	209	1363	58	125	1840	77	109
1875	2505	105	167	1817	76	164	2227	93	131
1876	1746	72	115	1591	66	142	2049	84	118
1877	1444	59	94	1504	61	133	2070	84	119
1878	1415	57	91	1426	57	124	1994	80	114
1879	1464	58	92	1356	53	116	2059	81	116
1880	1659	64	103	1902	74	160	1452	56	80
Mean of 24 years, 1855-78	1376	63	100	1017	46	100	1560	70*	100

* Mean of 26 years, 1856-80.

TABLE VIII.—ENGLAND AND WALES. DEATHS AND DEATH-RATES.

Twenty Years, 1881-1900. [Compare Diagram B.]

Year.	Scarlatina.			Septicæmia, etc.			Erysipelas.			Puerperal Fever.			Rheumatic Fever, etc.			Residual Rheumatism.		
	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.
1881	14,275	548	221	789*	30*	147*	2314	88.8	162	2287	87.8	115	2429	93.3	105	967	37.1	116
1882	13,732	521	210	684	26	127	2435	92.5	168	2564	97.4	127	2590	98.3	110	930	35.3	111
1883	12,649	475	191	608	23	112	2166	81.3	148	2616	98.2	129	2357	88.5	99	1015	38.1	120
1884	10,863	403	162	712	26	127	2145	79.7	145	2468	91.7	120	2714	100.8	113	873	32.4	102
1885	6,355	233	94	647	24	117	1996	73.3	133	2420	88.9	116	2912	107.0	120	868	31.9	100
1886	5,986	217	87	516	19	93	1523	55.3	101	2078	75.5	99	2530	91.9	102	867	31.5	99
1887	7,859	282	114	484	17	83	1853	66.6	121	2450	88.0	115	2650	95.2	107	981	35.3	111
1888	6,368	226	91	546	19	93	1635	58.1	106	2386	84.8	111	2697	95.9	107	942	33.5	105
1889	6,698	235	95	471	17	83	1209	42.5	77	1852	65.1	85	2258	79.4	89	897	31.5	99
1890	6,974	242	97	532	18	88	1375	47.8	87	1956	68.0	89	2418	84.1	94	938	32.6	102
1891	4,959	170	68	478	16	78	1263	43.4	79	1973	67.8	89	2547	87.6	98	1067	36.7	115
1892	5.618	191	77	529	18	88	1474	50.1	91	2356	80.1	105	2523	85.7	96	1029	35.0	110
1893	6,982	235	95	669	22	108	1921	64.5	117	3023	101.6	133	3085	103.7	116	878	29.5	93
1894	5,032	167	67	567	19	93	1353	44.9	82	2167	72.0	94	2646	87.9	98	799	26.5	83
1895	4,532	149	60	521	17	83	1099	36.1	66	1849	60.7	79	2257	74.1	83	888	29.2	92
1896	5,476	178	72	573	19	93	1101	35.7	65	2053	66.7	87	2677	86.9	97	850	27.6	87
1897	4,577	147	59	577	19	93	1014	32.5	59	1836	58.9	77	2527	81.1	91	914	29.3	92
1898	3,548	113	46	508	16	78	970	30.8	56	1707	54.2	71	2415	76.6	86	905	28.4	89
1899	3,722	115	46	655	21	103	1277	38.0	69	1908	59.2	77	2647	82.1	92	923	28.6	90
1900	3,844	118	48	735	23	112	1277	37.6	68	2017	61.8	81	2787	85.4	96	894	27.4	86
Mean	**7.003**	**248**	**100**	**590**	**20.5**	**100**	**1565**	**55.0**	**100**	**2198**	**76.4**	**100**	**2583**	**89.3**	**100**	**991**	**31.9**	**100**

* Owing to an alteration in the case of deaths from septicæmia following accidents, only approximate figures can be given for this year.

TABLE IX.—ENGLAND AND WALES.

Deaths and Death-Rates.

[Compare Diagram B.]

Year.	Enteric Fever.			Diphtheria (including *Cynanche Maligna*).		
	Annual Deaths.	Rate per Million.	Per Cent. of Mean.	Annual Deaths.	Rate per Million.	Per Cent. of Mean.
1856	...	...	...	603	32	17
1857	...	...	...	1583	82	43
1858	...	...	...	6606	339	178
1859	...	..	...	10184	517	271
1860	...	...	...	5212	262	137
1861	...	...	...	4517	224	118
1862	...	...	...	4903	241	126
1863	...	...	...	6507	316	166
1864	...	...	...	5464	262	137
1865	...	...	...	4145	196	103
1866	...	...	...	3000	141	74
1867	.	...	...	2600	120	63
1868	...	...	...	3013	138	72
1869	8660	390	161	2606	117	61
1870	8731	388	160	2699	120	63
1871	8461	371	153	2525	111	58
1872	8741	377	155	2152	93	49
1873	8793	376	155	2531	108	57
1874	8861	374	154	3560	150	79
1875	8913	371	153	3415	142	75
1876	7550	309	127	3151	130	68
1877	6879	279	115	2731	112	59
1878	7652	306	126	3498	140	73
1879	5860	236	97	3053	120	63
1880	6710	262	108	2810	109	57
1881	5529	212	87	3153	121	63
1882	6036	229	94	3992	152	80
1883	6078	228	94	4218	158	83
1884	6380	236	97	5020	186	98
1885	4765	175	74	4471	164	86
1886	5061	184	76	4098	149	78
1887	5155	185	76	4443	160	84
1888	4848	172	71	4815	171	90
1889	5011	176	73	5368	189	99
1890	5146	179	74	5150	179	94
1891	4875	168	69	5036	173	91
1892	4037	137	56	6552	222	116
1893	6801	229	94	9466	318	167
1894	4781	159	66	8774	291	153
1895	5326	175	72	7895	259	136
1896	5125	166	68	8993	291	153
1897	4851	156	64	7654	246	129
1898	5708	181	75	7661	243	128
1899	6304	198	82	9295	292	153
1900	5591	173	71	9345	290	152
Mean of 32 Years	6351	243	100	—	—	—
Mean of 45 Years	...	...	...	4854	191	100

TABLE X.—RAIN AT GREENWICH.

25 *Years*, 1856-81.

Year.	Rainfall.		Rainy Days.		$\frac{A + B}{2}$
	Inches.	Per Cent. of Mean. A.	Days.	Per Cent. of Mean. B.	
1856	21.5	89	144	95	92
1857	21.4	88	129	85	87
1858	16.8	69	112	74	72
1859	25.9	107	148	97	102
1860	31.6	130	192	126	128
1861	20.8	86	146	96	91
1862	26.5	109	179	118	113
1863	20.0	82	131	86	84
1864	17.2	71	112	74	72
1865	29.0	119	136	90	105
1866	30.5	126	184	121	123
1867	28.6	118	163	107	113
1868	25.3	104	143	94	99
1869	24.0	99	147	97	98
1870	18.5	76	128	84	80
1871	22.3	92	164	108	100
1872	30.0	124	197	130	127
1873	23.4	96	144	95	96
1874	20.0	82	139	91	87
1875	28.2	116	149	98	107
1876	24.2	100	168	111	105
1877	26.9	111	183	120	116
1878	29.2	120	167	110	115
1879	31.3	129	179	118	124
1880	29.8	123	152	100	112
Mean of 24 years, 1855-78	24.3	100	152	100	100

TABLE XI.—RAIN AT THREE TYPICAL STATIONS, 1881-1890.

Year.	Greenwich.					Wolverhampton.					Leeds.				
	Fall in Inches	Per Cent. of Mean. A.	Rainy Days.	Per Cent of Mean. B.	A + B / 2	Fall in Inches.	Per Cent. of Mean. A.	Rainy Days.	Per Cent. of Mean. B.	A + B / 2	Fall in Inches	Per Cent. of Mean. A.	Rainy Days.	Per Cent. of Mean. B.	A + B / 2
1881	25.2	111	154	95	103	25.70	99	155	99	99	28.64	114	180	103	109
1882	25.2	111	179	110	111	40.03	154	195	124	139	29.46	117	197	113	115
1883	21.9	97	173	107	102	29.44	113	173	110	112	29.81	119	170	98	109
1884	18.1	80	150	92	86	23.80	91	145	92	92	22.97	91	166	95	93
1885	24.0	106	166	102	104	26.93	103	173	110	107	21.68	86	178	102	94
1886	24.2	107	163	100	104	33.38	128	179	114	121	25.00*	100*	180*	103*	102*
1887	19.9	88	142	88	88	22.96	88	139	88	88	16.54	66	159	91	79
1888	27.5	121	167	103	112	24.80	95	173	110	103	23.38	93	163	94	94
1889	23.3	103	159	98	101	34.75	133	146	93	113	20.98	84	152	87	86
1890	21.9	97	162	100	99	21.18	81	148	94	88	23.02	92	171	98	95
1891	25.1	111	178	110	111	27.00*	104*	156*	99*	102*	25.12	100	200	115	108
1892	22.3	98	170	105	102	26.76	103	156	99	101	27.88	111	193	111	111
1893	20.1	89	154	95	92	19.49	75	139	88	82	22.94	91	168	96	94
1894	26.9	119	192	118	119	23.88	92	167	106	99	28.10	112	193	111	112
1895	19.7	87	158	97	92	22.55	87	138	88	88	30.44	121	181	104	113
1896	22.4	99	161	99	99	21.01	81	165	105	93	24.78	99	169	97	98
1897	22.1	98	169	104	101	23.78	91	159	101	96	25.49	101	177	102	102
1898	18.9	83	142	88	86	21.01	81	137	87	84	22.47	89	159	91	90
1899	22.3	98	141	87	93	25·87	99	133	85	92	23.32	93	151	87	90
1900	22.3	98	165	102	100	29.99	104	166	106	105	30.28	121	179	103	112
Mean of 20 Years	22 7	100	162	100	100	26.07	100	157	100	100	25.12	100	174	100	100

* Owing to a gap in the record, the mean of neighbouring stations is given.

TABLE XII.—SEASONAL RAINFALL AT GREENWICH, 1881-1900.

Year.	First Four Months: JANUARY TO APRIL.					Second Four Months: MAY TO AUGUST.					Third Four Months: SEPTEMBER TO DECEMBER.				
	Fall in Inches	Per Cent. of Mean, A.	Rainy Days.	Per Cent. of Mean, B.	$\frac{A + B}{2}$	Fall in Inches	Per Cent. of Mean. A.	Rainy Days.	Per Cent. of Mean, B.	$\frac{A + B}{2}$	Fall in Inches	Per Cent. of Mean. A.	Rainy Days.	Per Cent. of Mean B.	$\frac{A + B}{2}$
1881	6.1	100	46	86	93	9.5	122	49	97	110	9.7	110	59	101	106
1882	6.0	99	42	79	89	7.3	94	64	127	111	11.8	134	73	124	129
1883	7.1	117	58	109	113	5.8	75	48	95	85	9.1	103	67	114	109
1884	5.8	95	54	101	98	5.6	72	42	83	78	6.7	76	54	92	84
1885	7.3	120	51	96	108	5.6	72	41	82	77	11.1	126	74	126	126
1886	6.6	109	59	111	110	8.3	107	47	93	100	9.3	105	57	97	101
1887	4.8	79	39	73	76	6.6	85	40	80	83	8.5	96	63	107	102
1888	6.1	100	61	114	107	14.5	186	60	119	153	7.0	79	46	78	79
1889	6.2	102	62	116	109	9.3	120	51	101	111	7.8	88	46	78	83
1890	6.9	113	56	106	110	10.9	140	63	125	133	4.1	46	43	73	60
1891	4.5	74	46	86	80	10.8	139	68	135	137	9.8	111	64	109	110
1892	4.6	76	52	98	87	8.5	109	53	105	107	9.2	104	65	111	108
1893	4.7	77	47	88	83	5.9	76	46	91	84	9.5	108	61	104	106
1894	6.8	112	59	111	112	9.9	127	70	139	133	10.2	116	63	107	112
1895	4.5	74	54	101	88	6.2	80	45	89	85	9.0	102	59	101	102
1896	4.6	76	47	88	82	5.3	68	41	82	75	12.5	142	73	124	133
1897	9.0	148	64	120	134	6.8	87	50	99	93	6.4	72	55	94	83
1898	4.2	69	44	83	76	6.6	85	53	105	95	8.1	92	45	77	85
1899	8.1	133	60	113	123	4.5	58	32	64	61	9.8	111	49	83	97
1900	7.7	127	65	122	125	7.6	98	43	85	92	7.0	79	57	97	88
Mean of 20 years	6.08	100	53.3	100	100	7.78	100	50.3	100	100	8.83	100	58.7	100	100

TABLE XIII.—RHEUMATIC FEVER IN THREE REGIONS OF ENGLAND AND WALES.

1881 TO 1900.

—	South-Eastern Region.			Western and Midland Region.			Northern Region.		
	Deaths.	Rate per Million.	Per Cent. of Mean.	Deaths.	Rate per Million.	Per Cent. of Mean.	Deaths.	Rate per Million.	Per Cent. of Mean.
1881	944	102.1	118	665	82.1	98	820	95.0	98
1882	909	97.2	111	793	97.1	116	888	101.7	105
1883	821	86.6	99	690	83.9	100	846	95.7	98
1884	997	103.8	119	746	90.0	107	971	108.5	112
1885	1044	107.3	123	833	99.7	119	1035	114.3	118
1886	867	87.9	100	721	85.7	102	942	102.7	106
1887	932	93.3	107	717	84.5	101	1001	107.9	111
1888	912	90.1	103	812	95.0	113	973	103.6	107
1889	755	73.7	84	626	72.7	87	877	92.2	95
1890	907	87.4	100	629	72.5	87	882	91.6	94
1891	911	86.6	99	705	80.6	96	931	95.6	98
1892	873	82.0	94	749	84.8	101	901	91.4	94
1893	1167	108.2	124	879	98.6	118	1039	104.2	107
1894	880	80.6	92	769	85.4	102	997	98.8	102
1895	724	65.5	75	665	73.1	87	868	85.0	87
1896	933	83.3	95	777	84.6	101	967	93.6	96
1897	887	78.2	89	679	73.2	87	961	91.9	95
1898	826	71.9	81	682	72.8	87	907	85.7	88
1899	948	81.5	93	735	77.7	93	964	90.0	93
1900	970	82.4	94	785	82.2	98	1032	95.3	98
Mean of 20 years.	910.4	87.48	100	732.9	83.8	100	940.1	97.2	100

DISCUSSION.

THE PRESIDENT: We have listened to-night to a Paper of very singular interest, and I am sure you will have a great deal to say in the way of observations upon it. Before I ask Sir William Church to open the discussion, I will ask Dr. Bulstrode to read a letter which he has received from Professor Kenwood, who is unable to be present to-night.

DR. BULSTRODE: Professor Kenwood writes from University College—It is with great regret that I find myself unable to attend the meeting, but I have read with great interest the valuable Paper by Dr. Longstaff, which constitutes the programme for this evening. There is just one item in the Paper upon which I have thought that I could send a few lines which may be of interest to Dr. Longstaff and the meeting. At the commencement of the Paper he draws attention to the practically-identical character of the mortality curves of erysipelas and puerperal fever; and he adds that there appears to be some kind of inverse relationship between erysipelas (as with certain other diseases) and the amount of rainfall.

Some five years ago, Dr. A. Griffith and I collected together much material for the purpose of an inquiry into the circumstances accounting for the unequal incidence of erysipelas upon different London areas, and to this end we collected all the facts relating to the notification of this disease in the Metropolitan areas for several years. We concluded that the amount of sickness notified in relation to such a disease as erysipelas was likely to prove of greater value to us, for the purpose of our special inquiry, than mortality returns. The result of this work, which has never hitherto been published, supports Dr. Longstaff's conclusion: for the curves of erysipelas and puerperal fever were practically identical in the Metropolis—whether they be sickness or mortality curves. We did not, however, satisfy ourselves that the facts warranted the conclusion that there was a definite relationship existing between any meteorological circumstances and erysipelas incidence *in the Metropolis*. The considerable variations in the incidence of the disease in various parts of the Metropolitan area during the same week or month seemed to support this view, and we did not find that the erysipelas notification curves, drawn for the different weeks of several years, gave

Erysipelas and Meteorological Data during the Ten Years 1888 *to* 1897 (*London*).

Year.	1888.	1889.	1890.	1891.	1892.	1893.	1894.	1895.	1896.	1897.
Death-rate per 1,000 ...	0.06	0.04	0.06	0.05	0.07	0.10	0.05	0.04	0.05	0.04
Range of the highest and lowest T° :—										
January	28.7	31.1	29.5	36.1	29.2	37.3	38.9	34.7	24.7	23.6
February	32.9	37.7	22.2	37.0	35.5	31.9	32.5	38.9	32.2	30.1
March	30.7	40.4	50.6	32.0	37.5	41.7	37.2	40.0	35.8	34.9
April	37.1	31.5	33.8	38.2	44.8	47.3	38.8	38.0	24.7	39.5
May	40.9	38.5	38.5	49.5	56.3	40.4	38.5	50.5	42.8	44.1
June	40.8	37.6	37.4	34.1	44.1	52.1	40.1	41.7	44.0	44.5
July	29.9	34.8	33.8	39.7	35.4	43.4	39.3	34.3	41.0	39.6
August	40.7	40.2	39.3	32.6	39.0	48.8	30.9	35.3	31.7	40.3
September	30.3	45.2	38.1	37.1	37.2	46.3	34.5	40.2	32.3	32.5
October	40.6	25.1	44.7	36.2	31.2	35.4	30.9	47.8	33.7	35.3
November	25.3	32.5	37.3	28.2	30.0	32.0	32.6	31.5	24.9	31.1
December	33.0	30.4	28.8	40.6	37.8	37.7	25.4	30.3	25.2	32.2
Means—12 Months ...	34.24	35.42	36.17	36.76	38.16	41.18	34.96	38.60	32.83	35.64
Relative Humidity :—										
January	91	89	87	88	89	89	87	88	89	93
February	86	86	87	89	89	87	83	76	88	86
March	84	80	80	80	83	77	81	84	83	81
April	78	81	83	73	69	65	76	77	71	78
May	68	79	68	80	65	67	77	68	66	67
June	76	74	73	71	70	65	74	62	71	74
July	82	73	74	74	71	70	74	63	65	68
August	79	75	78	79	74	66	88	75	83	73
September	80	75	80	77	74	71	79	86	82	77
October	84	88	80	84	84	80	85	84	84	83
November	87	88	89	88	90	85	87	88	85	88
December	91	89	86	88	85	86	86	87	87	87
Means—12 Months ...	82.2	81.4	80.4	80.9	78.6	75.7	81.4	78.2	79.5	79.6

Year.	1888.	1889.	1890.	1891.	1902.	1893.	1894.	1895.	1896.	1897.
Death-rate per 1,000 ...	**0.06**	**0.04**	**0.06**	**0.05**	**0.07**	**0.10**	**0.05**	**0.04**	**0.05**	**0.04**
Range of the Means of highest and lowest T°:—										
January	9.5	9.4	11.0	10.5	10.6	9.1	10.6	9.0	8.9	8.1
February	8.7	11.6	10.2	13.5	10.8	11.2	12.3	13.6	11.5	9.4
March	11.0	14.4	14.6	13.2	13.9	20.3	18.2	15.4	13.3	13.1
April	15.3	14.9	16.2	17.1	**22.8**	**25.2**	**18.7**	17.1	17.1	15.7
May	**19.9**	16.9	**19.7**	**17.5**	**21.0**	**22.8**	**18.6**	**22.3**	**22.0**	**20.5**
June	**17.3**	**19.3**	**18.1**	**19.3**	**20.0**	**23.5**	**18.3**	**23.0**	**21.2**	18.7
July	14.8	**17.0**	16.7	**17.8**	18.3	19.3	18.2	18.1	**22.3**	**20.5**
August	**17.2**	**17.9**	17.4	10.5	18.9	20.7	15.9	18.0	18.3	**18.8**
September	15.2	16.7	**18.8**	17.1	16.7	18.8	15.0	**22.2**	13.4	15.5
October	16.5	12.8	15.5	13.3	13.6	14.4	11.7	13.6	13.1	14.1
November	7.7	9.5	11.4	10.3	9.6	11.9	10.5	10.9	11.1	10.7
December	10.0	9.8	8.5	11.0	10.0	11.4	10.3	9.7	10.2	9.8
Means—12 Months ...	**13.62**	**14.18**	**14.84**	**14.20**	**15.51**	**17.40**	**14.86**	**16.07**	**15.20**	**14.57**
Rainfall :—										
January	0.90	0.81	2.46	1.80	1.50	1.44	2.87	1.96	0.78	2.05
February	0.78	2.28	1.05	0.01	1.62	**2.87**	1.75	0.12	0.30	**2.75**
March	3.34	1.36	1.76	2.01	1.04	0.32	1.19	1.42	**3.20**	**3.42**
April	2.37	2.06	2.02	1.13	0.99	0.24	1.74	1.35	0.55	1.57
May	1.18	**3.22**	1.25	2.72	1.51	0.80	1.85	0.34	0.14	1.08
June	2.31	2.03	**2.82**	0.86	2.46	0.74	1.84	0.30	2.27	1.87
July	**4.91**	**2.64**	**4.20**	**3.82**	1.62	**2.46**	**3.25**	**3.41**	1.03	0.64
August	**3.61**	1.80	**2.59**	**4.75**	**3.06**	1.61	2.85	**3.09**	1.92	**2.92**
September	1.43	1.77	0.70	1.03	2.12	1.07	1.05	1.28	**5.51**	**2.75**
October	1.23	**3.75**	1.20	**4.80**	**3.78**	**3.87**	**4.45**	2.84	3.05	0.56
November	**4.38**	0.89	1.62	1.98	**2.53**	2.17	2.85	**3.17**	1.17	1.04
December	1.29	1.23	0.68	3.24	1.37	2.23	2.28	2.19	**3.61**	2.20
Means—12 Months ...	**2.31**	**1.98**	**1.86**	**2.35**	**1.97**	**1.65**	**2.33**	**1.71**	**1.96**	**1.90**

indication of any relationship between incidence and rainfall. While during April, May, and June the incidence of erysipelas is at its lowest, the rainfall for that quarter is generally exceeded by that of other quarters during the ten years shown on the accompanying Table. November, December, January, and February are the months during which the relative humidity of the atmosphere is at its highest, whereas erysipelas incidence in the Metropolis increases in September and October, and a rapid decline sets in through January and February. On the other hand, Dr. Longstaff's conclusion is supported by the fact that during the year 1893, a year of exceptionally high erysipelas incidence in the Metropolis, the relative humidity and rainfall were the lowest recorded during the ten years ending 1897. In conclusion, I may further state that we found it *very rare* for a case of puerperal fever to be preceded by a recent case of erysipelas; and we concluded that the identity of the curves was best explained on the ground that the same circumstances and conditions are favourable to the appearance of both diseases, and that puerperal fever rarely (if ever) occurs in consequence of direct infection from erysipelas.

SIR WILLIAM CHURCH (President of the Royal College of Physicians): You have called upon me to open this discussion, which I think is rather hard upon me. I came here to-night as a guest, and really I had not thought of taking any part in the discussion. I felt certain that I should be greatly interested in this subject, which has had an attraction for me almost all my medical life. I was also quite sure that I should hear a most interesting Paper, and I have to thank Dr. Longstaff, in the names of all who have heard it, for the extremely suggestive and able Paper that he has placed before us. We have also the advantage of having these diagrams, which enable us to follow the whole of the Paper with great ease.

I feel myself specially incompetent to speak upon this Paper—or, I might say, on any Paper of this nature. Whether or not there is something wanting in me I don't know, but I have always had a great dislike of statistics, and I have always had the greatest possible distrust of them. I have not the slightest distrust of Dr. Longstaff, but I have the greatest possible distrust of statistics, and I venture to say that, without the slightest wish or intention to detract from the value of them as far as

they go, or of the labour and care with which they are put together.

It so happens that in another position—in a certain public position—I have had occasion to make a little inquiry into the value of medical certificates, and although I am wandering a little from the subject, Dr. Newsholme will uphold me when I say that you would be astonished if you knew the amount of error which is made by the very highest trained medical men in recording the causes of death. In the statistical investigations we have tried to make with regard to the incidence of cancer, we have found that the amount of error in the Returns is very much greater than anyone would suppose. The probable error is shown in this way: We have taken the trouble to get statistics of "complete" cases of cancer (by "complete" cases I mean cases of cancer in which the patient has, after death, been submitted to a post-mortem examination). In these the amount of error is very large; and if it were possible to follow up those cases in which cancer is not available to direct examination, it would be still larger. It is probably the case that medical diagnosis is not very correct in most other diseases.

One thing has struck me very much, speaking as a practical and hospital physician. Now, here is a whole series or class of diseases put down under the head of "Residual Rheumatism." I do not understand what residual rheumatism includes in the slightest. Rheumatism, apart from pericarditis or acute inflammatory rheumatism of the heart or lungs, is not a fatal disease; and with the exception of a small number of cases of hyper-pyrexia, acute rheumatism is not a fatal disease. Therefore, deaths that are due to residual rheumatism I imagine probably include a very large group, and a very wide group, many of which are due to senility and other causes. I fancy they have been put down as rheumatism on the death certificate when there has been no accurate knowledge of what death has been due to. Personally, I do not understand the group of diseases which is placed in the column "Residual Rheumatism" in the Registrar-General's Returns.

I have paid some attention to rheumatism, or rheumatic fever, which I hold to be an entirely distinct disease from what is spoken of as rheumatism. We know that if we take unusual exercise we are the next day extremely stiff. Now, that sort of stiffness, or pain very similar to that, is very often called, by the patient and by the doctor, rheumatism. I have had some experience in military

hospitals and camps, and I have noticed that a large number of men come into the hospitals suffering from what is called rheumatism, especially when they are going through campaigns or any great stress of labour, but the majority of them appear to me really to be suffering rather from the effects of fatigue than from any direct disease.

Then there was another point in Dr. Longstaff's Paper which interested me very greatly: he has not told us anything at all of the probable cause, that does not come within the scope of his Paper; but acute rheumatism, or rheumatic fever, is an urban disease and not a rural disease; and he remarks that rheumatism has gone up in South Wales, where there are large towns such as Cardiff, which has increased enormously in the years he has taken. He mentions also that Yorkshire is another place where it has rather increased. And here again you have a large number of manufacturing towns.

As regards the allusion to Dr. Newsholme's work, I think there can be no doubt that rheumatism is associated with certain houses, but I cannot agree with Dr. Newsholme with regard to the condition of the subsoil having much to do with it, because rheumatism is particularly a disease of towns, where you have a large area built upon, drained, paved, and tunnelled, and there can be very little subsoil water. Almost all the water in the London area runs off the surface at once.

I think the view is now generally accepted that rheumatism is due to an organic germ of some sort, but there is no disease in which there has been greater clinical confusion than in rheumatism and acute rheumatism. If you read the accounts of the great French physicians who described it, you can have no doubt that many of their cases were really cases of pyæmia. In the accounts of their post-mortems of the fatal cases, not only was the pericarditis purulent—which true rheumatism rarely produces—but a large number of them had purulent effusions into the pleuræ. Continental physicians have always talked about pleurisy of rheumatic origin, which is a very unusual thing in this country. It is very seldom I have met with cases in which there has been pleurisy. You get cases in which there is pneumonia, and some of these may possibly be fatal: although one thing that is remarkable is that, however much acute pneumonia you may have present in rheumatic fever, recovery generally follows.

Therefore, I say, with regard to the statistical examination of diseases like rheumatism, it is most difficult to

accept the conclusions drawn. It is not like fevers, in which, although there is a possibility of error, it is not great—it is so very great in rheumatism. It is not so great in erysipelas, which is a more marked disease. Of course, we know there was great error at one time, before typhus and typhoid fever were well separated, but there is now little room for error in typhus fever, although plenty of room for error in typhoid: but still not so great room as in the various forms of disease which have in the past been confounded together as acute rheumatism.

Then, I think that all physicians who have had much practice are convinced that there is some sort of connection between what is spoken of here as "rheumatic arthritis" and, I do not say puerperal fever, but a toxic condition which very frequently does arise from the uterus. I have met with many cases, and I think all physicians agree that a good many of those distressing cases of rheumatoid arthritis that one meets with in women occur in those who have at some time—a few months before, or even a few years before—suffered from some uterine trouble or discharge, and that seems to be the point from which it started.

Then, again, there can be no doubt you have added on to the effects of an attack of rheumatic fever on the valves of the heart—another cause of death. I cannot think that ulcerative endocarditis is only another stage of the same organism which produces rheumatic fever. What the organism is that produces ulcerative endocarditis I cannot say, but I think it is a distinct organism from that which produces rheumatic fever. I have myself had several cases in which one has got perfectly pure cultures of *pneumococcus* from cases of ulcerative endocarditis; and further, I think the rheumatic fever produces a condition of the valves which becomes a *nidus* for another organism. That is, I think, the connection that exists between ulcerative endocarditis and rheumatism, which I look upon as being distinct diseases, and due to distinct causes.

I am afraid I have not been able to make any remarks from the proper statistical point of view. I trust the crudeness of these remarks will be pardoned, as I did not come here prepared to make any remarks whatever.

I am glad that Dr. Longstaff does not think that rheumatism is a disease connected with damp and cold. I went at one time very closely into the Army statistics, although, as I say, I distrust all statistics. I went very closely into them because I thought, Here you have a series of men who

have lived more or less under the same conditions in very different climates, and they are all men at just about the most vulnerable time of life for rheumatic fever (or perhaps a little past it, as from 15 to 25 is the most vulnerable time). I went very closely into these statistics, and I found that India has a higher percentage of admissions to hospital for rheumatism than Ireland. Ireland, as we know, has a climate which is supposed to be damp, yet the percentage of men invalided from rheumatism is much lower than in England, especially in the Midland area. Scotland, again, has less than England, and I found that the north-east coast of England is freer from rheumatism than is the west coast. In those points I am glad to see that Dr. Longstaff's figures support me.

It seems to me that rheumatism is one of those diseases about which it is most difficult to arrive at a conclusion from statistics.

DR. NEWSHOLME: Mr. President and Gentlemen,—I think we shall all agree as to the great value of Dr. Longstaff's Paper, and I am sure we shall all join in congratulating him on the exceptional fact that at the end of twenty-five years he is able to bring up to date the conclusions which he stated in the year 1880.

These conclusions have necessarily been quoted by every subsequent worker on this disease and allied diseases, and will continue to be quoted.

In speaking on the Paper, I must necessarily take up a somewhat critical attitude, but although that must be so, I am sure Dr. Longstaff will not think I am merely carping: any more than the meteorologist can be said to be carping at the sun because he gives his attention chiefly to sun-spots.

I must agree with what Sir William Church has said—that rheumatism is a disease concerning which statistics can mislead us. Of course, I refer chiefly to death statistics.

I am glad to see that one of the points Dr. Longstaff has gone into confirms my own opinion, that the national death statistics of rheumatism, in view of the see-sawing which has taken place in the classification of deaths from this cause, cannot be trusted to any very great extent.

I am bound also to agree partially with Sir William Church, though not entirely, as to the fallacy due to ulcerative endocarditis. I do not think Dr. Longstaff has completely met Sir William Church's objection with regard

to this disease. Dr. Longstaff says that before ulcerative endocarditis was entered separately, it was entered when recognised under the head of valvular diseases, and not under rheumatism; but that does not meet the point. In the past it was not recognised, and the point is: What was it entered under in those days? Quite probably under "Rheumatism of the Heart;" and I think there has been confusion in the past between rheumatism of the heart and infective or ulcerative endocarditis.

I do not think that Dr. Longstaff's curves have the same value in relation to rheumatism as they have in relation to scarlet fever, puerperal fever, and erysipelas; because in his figures only the fatal cases are included, and the fatality in scarlet fever (until recently), and in puerperal fever and erysipelas, is much greater than in rheumatic fever. I have myself shown by statistics that the fatality in cases of rheumatic fever was only about 2 per cent. in Norway, where cases of rheumatic fever are compulsorily notified; and Dr. Southey found that the fatality was only 1.12 per cent. in a long series of cases at St. Bartholomew's Hospital. It is clear, therefore, that we have to deal here with a disease the fatality in which is very low, and thus the mortality results must give a very imperfect idea of the prevalence of the disease. In the case of puerperal fever, the death returns form a much better index of the prevalence of the disease than notifications. You will realise this more especially when I tell you that in my own town the apparent fatality of puerperal fever in some years is as much as 150 per cent., owing to imperfect notification!

A further reason for the lack of complete trust when one has to deal with rheumatism statistics is the fact that there has been discovered in recent years a remedy—Salicin, with its derivates—which, although not exactly a specific, is very nearly one. Owing to this fact, it is probable that there are now less deaths from rheumatism for a given number of cases than formerly, with the result that something has to added to the mortality curve now as compared with the past. Until we get a general notification of cases of rheumatic fever, I do not think there is any possibility of our being able to completely investigate the natural history of this disease, which, in my opinion, is scarcely second in importance to consumption itself.

My third objection is that Dr. Longstaff's curve deals with the whole of England and Wales. It would have been better, I think, if he had given it for London alone; because, by giving a curve for the whole country together

one tends to fuse the individual epidemic peaks for different parts of the country, which do not necessarily occur in the same year in all parts of the country. For instance, take the curve for London, and compare it with the curve for England and Wales as a whole. The maximum number of deaths from rheumatic fever in London alone occurred in 1868, whereas the maximum number for the whole country occurred in 1869, and similarly the next maximum occurred in 1874 in London, and in 1875 in the country as a whole. I was able to show (and, I think, conclusively) by hospital records in London that the maxima in the big epidemic of 1874 to 1876 were reached in different parts of London in different years. The maximum number of cases at University College, for instance, was reached in 1876, two years later than that of some other parts of London. Similarly in the case of diphtheria, different boroughs in London had their maxima in different years; and some of them suffered from relapses of epidemic diphtheria just as individuals suffer from relapse of personal infection. The same thing is shown in the statistics of Norway. The statistics of Christiania differ from those for the whole of Norway, the maximum number of cases of rheumatic fever being reached at an earlier year in the capital.

For these various reasons I do not think death statistics altogether trustworthy for rheumatic fever.

I may point out that the county death-rates given in Dr. Longstaff's Paper are crude death-rates, which the Registrar-General has largely abandoned in his reports, and that is very important. Also, Dr. Longstaff has not obtained some of the lessons which might have been obtained if he had given each county separately, instead of taking larger divisions. He classes together the whole of Yorkshire. His main hypothesis has been that the amount of rheumatism has been inverse to the annual rainfall. That being so, is he likely to bring it out by classing together the East and North Ridings, which are comparatively dry, with the West Riding, where the rainfall is excessive? In my statistics, taking rheumatic fever in the whole country as 100, in the West Riding the corrected death-rate was 114, in the East Riding, 85, which is a very marked difference.

Then, again, I think that an average death-rate for a series of years is open to objection when dealing with an epidemic disease. Such a method tends to behead the peaks of the epidemics, and to elevate the valleys, and generally to conceal the true incidence of the disease. It is true that thus one gets larger data to deal with, but one loses

the epidemic incidence of the disease. (I am now referring to geographical distribution, in connection with which alone Dr. Longstaff uses this method.)

Although the death-rates do bring out many points, which Dr. Longstaff has taken full advantage of, what is to be learnt about rheumatic fever cannot be learnt from death returns alone. I have shown that epidemics of puerperal fever and erysipelas are much less protracted than those of rheumatic fever. This is particularly well shown in the Norwegian figures. The smallest epidemic of rheumatic fever corresponds with the largest epidemic of erysipelas, and *vice versâ*. It is shown also in the Berlin figures, which I gave in the Milroy Lectures.

Another important thing is brought out by cases as distinct from deaths. I worked out the statistics of 62,834 cases of rheumatic fever and 41,537 cases of erysipelas in Norway. In erysipelas the percentage of total cases occurring under fifteen years of age was 15.1, and in rheumatic fever, 15.3. I think that is a striking proof of, if not actual identity, a close connection between these two diseases, which is one of Dr. Longstaff's main points.

In view of these striking resemblances, it is not surprising that some of the old clinicians properly regarded rheumatic fever as an epidemic disease. Trousseau illustrated this in his work; and the greatest statistician of the last century—I refer, of course, to Dr. Farr—in his fourth Annual Report, definitely and distinctly classified rheumatic fever among the zymotic diseases; but the Royal College of Physicians, in their nomenclature, relegated it to a place between pellagra and gout, where it still remains.

I have referred to the necessity of taking case-rates instead of death-rates. I will take scarlet fever as an instance. I venture to suggest that if Dr. Longstaff had taken Sir Shirley Murphy's curve of cases for the last ten or fifteen years in London, he would have found a closer approach to an inverse relationship between scarlet fever and rainfall than is shown when only the death-rates are taken. Some unknown cyclical factor has diminished the death-rate of scarlet fever, which has not lowered the case-rate to the same extent; and it is necessary, therefore, to work in this instance with cases.

Then, again, I should like to point out that in his chief curve, Dr. Longstaff has taken the rainfall of Greenwich, and compared it with the rheumatism mortality of the whole country. In other cases he compares the rheumatism

of certain divisions of the country with the rainfall in selected towns in these divisions. As already seen in the case of Yorkshire, it is doubtful if this does not partially conceal the point to be elucidated.

I venture to refer to one personal point. Dr. Longstaff quotes me as saying that rheumatic fever is "essentially a soil disease," and adds that I "therein compared it with malaria." I suggest that it would be more correct if he would delete the word "compare" and substitute the word "contrast." The main essence of my labours on this point was to *contrast* rheumatic fever and malaria: because although rheumatic fever and malaria are both in a sense soil diseases, they are the exact antithesis of each other, as regards the soil conditions in connection with which they occur. I may quote my own words in this respect (*Lancet*, p. 664, March 16th, 1895):—

"Alterations of drought and moisture with a a moderately high temperature seem most favourable to malaria; whereas rheumatic fever is most abundant when the deficiency of rainfall has been exceptionally protracted, and has produced a marked and exceptional drying of the subsoil."

May I refer to the dead who have done so much to advance our control over this disease, particularly to Dr. Maclagan, who argued that since rheumatism occurs (as he thought) generally in damp and low-lying places, the remedy was to be sought for in the same situations. He came to the conclusion that the willow would probably contain a remedy, and by following up this hypothesis, he actually obtained the remedy—Salicin—which has done such wonderful things in relieving suffering humanity. I wish that some distinguished physician would take up the subject of the discoveries of empirical medicine, and suitably immortalise those discoveries which, although based on imperfect—or even erroneous—pathology, have in some instances done more for the relief of suffering than has been achieved in other instances by scientific medicine.

It is now certain that the assumption on which Dr. Maclagan proceeded was not a correct one. It is equally certain that the statement which is still embodied in important text-books, that rheumatism evinces "a preference for damp and deeply-cleft valleys" is erroneous; but it is also true that on this erroneous basis has been built up one of the most important therapeutic cures which we possess.

I venture to allude to Sir William Church's views with regard to the non-relation of subsoil conditions to rheuma-

tic fever. I think he has misapprehended me. He has alluded to the fact that in London there can be no relationship between subsoil water-level and the prevalence of rheumatism, as I have contended, because, owing to drainage, tunnelling, etc., there can be little or no subsoil water under London. That may be so; no more is there under Christiania, for which place also I have given extensive statistics. But my contention is that the level of the subsoil water is merely an index, and I believe the most perfect index we have: much more so than the inverse rainfall. The same conditions may obtain in the absence of the subsoil water as in its presence: just as the works of a clock may still go on even if the hands are removed. The hands of the clock are represented by the subsoil water, which is a very convenient index; but the dry and warm condition of the soil favouring rheumatic fever may be there, even in the absence of the index.

I have never committed myself to a purely miasmatic theory of rheumatic fever, but have contended that a certain stage of the life-history of the rheumatic fever organism is passed in the soil. I can best summarise my views by the following quotation from a contribution in the *Practitioner* for January, 1901:—

"It seems most feasible to regard rheumatic fever as essentially a soil disease, due to a saprophytic soil organism, which is 'drowned out' in wet years, multiplies rapidly in dry years, and is transferred to the human recipient by unknown means. Possibly dust convection accounts for a large proportion of the cases. In dry years the number of contagious particles is probably increased, and the facility with which they are communicated to the human subject is similarly increased. In the light of our recently-acquired knowledge of the methods of infection in malaria, it is not perhaps too far-fetched to surmise that inoculation of the contagium of rheumatic fever may be caused by domestic vermin, or that the house-fly may convey it to milk or other foods. Perhaps bacteriology will in a few years be able to throw important light on what, for want of more accurate knowledge, can at present be only guessed at."

I am perfectly aware that both Dr. Longstaff's investigations and my own deal only with secondary causes—the real cause being some micro-organism. But in getting at the facts which will enable us to prevent the spread of a disease, it sometimes happens that we are able to discover and counteract the general conditions which favour the prevalence of the disease, more easily than to prevent the action of the micro-organism itself. Moreover, we cannot be said to know the natural history of any disease until we know not only the *causa causans*, but also those efficient causes, such as climatic conditions, in the absence of which the disease does not become epidemic.

DR. HAMER: Mr. President and Gentlemen,—There is only one point I should like to speak upon, and that is the measure of importance to be attached to the phenomenon upon which, in his characteristic manner, Dr. Longstaff lays the very smallest emphasis possible, namely, the correspondence between rainfall fluctuations and fluctuations in mortality from certain diseases in the later period of years. It does not seem to me remarkable that the correspondences should be less marked in the later period than they were in the earlier one. On the other hand, it would have been indeed remarkable if those correspondences had been as conspicuous in the latter as in the former.

What we have to recognise is this: I read, the other day, a statement made by a distinguished epidemiologist, to the effect that the powers of Nature are such that she does not permit phenomena to originate from isolated causes. Bearing that in mind, we have to recognise that these great waves of scarlatina mortality in the 'sixties and 'seventies were not entirely due to rainfall, or to any single cause of that kind, but were due to a number of causes, of which this meteorological cause was undoubtedly one. It is only rarely that the opportunity is afforded of seeing the effect of some particular force, and of thus being enabled to draw the conclusion that it is in operation. As the result of the inter-play of forces, there is, however, at rare intervals, thrown, as it were, into prominence, a set of facts, such as that with which Dr. Longstaff had to deal, and from which he was able to draw conclusions as to particular cause and effect relationships. Such a set of facts is cast up like wreckage along the shore, which tells in a most imperfect way of what has been happening in the mighty deep itself, and of the inter-play of forces that has there been going on.

Dr. Longstaff has pointed to the year 1893, in the second series of years, as being one in which there was a moderate-sized peak corresponding to low rainfall. There was something of the same kind in 1887; and if Dr. Longstaff's inquiry had extended to quite recent times, the year 1903 would, no doubt, have also been referred to, for in that exceptionally wet year the number of admissions to London fever hospitals fell to a marked extent.

DR. BOND: I do not know that I have anything of importance to say. I was struck, in Diagram C, with reference to the average weekly deaths in the different months. This diagram gives the average deaths from rheumatic

fever, erysipelas, and puerperal fever during the twenty years 1880 to 1901, and it shows there has been a greater number of deaths in the winter months than in the other months of the year. For instance, from October to February the numbers are highest. That seems to show that cold has some effect, because the winter months are the coldest in the year, and if you look at the peaks they are all in those months. The curves all rise above the mean in the month of October, and go down in the months of February or March, and remain below the mean until October.

DR. J. T. C. NASH: The value of Dr. Longstaff's Paper is emphasised by the criticism of Sir William Church and Dr. Newsholme.

I have no critical remarks to offer, but I should like to say that a point which interested me greatly is the support which Dr. Longstaff's researches appear to give to the doctrine of evolution as applied to pathogenic organisms and disease-processes.

Even in his original *Studies in Statistics*, so long ago as 1880, Dr. Longstaff could trace and demonstrate certain striking points of resemblance in the natural history of certain infective diseases, which can be easily differentiated when they present themselves as definite specific clinical types, answering to the names of erysipelas, puerperal fever, scarlet fever, and rheumatic fever. When, however, less typical cases present themselves, diagnosis between these various so-called "specific diseases" becomes less dogmatic.

What evidence bacteriology has to offer up to the present time in connection with these infective processes is in favour of Dr. Longstaff's conclusions, that erysipelas and puerperal fever "are both due to one poison." Similar evidence is available as regards scarlet fever and rheumatic fever.

Streptococci are the organisms which appear to be causally related to all of these diseases.

It may readily be conceived that the original organism, in the course of the vicissitudes of its descendants, met with varying evolutionary conditions of existence, giving rise to varieties and strains, some of which approach specificity, and tend to breed true fairly permanently; giving rise in turn to definite combinations of clinical symptoms sufficiently distinctive to form clinical entities. But all the time there may be a distinct relationship, as I have tried to describe it.

THE PRESIDENT: I must now ask Dr. Longstaff to reply. We have had a most interesting discussion, and I personally do not feel disposed to add more conundrums for him to answer, nor do I wish to anticipate what he will have to say in his reply.

My own feeling goes with Dr. Longstaff in the point to which criticism has been chiefly directed, and I am reminded of the dictum that while you can prove anything by statistics, you can prove nothing without them. I admit that there is considerable doubt as to the accuracy of many of the individual death certificates which form the basis of the Registrar-General's mortality statistics. But to assume that collectively they are untrustworthy, and that the failures in diagnosis or statement are fatal to any broad conclusions on a statistical basis, is a very different matter. The mortality graphs—of age, season, and yearly totals—which Dr. Longstaff has worked out, appear to me to be too characteristic and orderly to fail to carry conviction as to their substantial accuracy. Thus the age graph of fatal rheumatism, after removing from that vague class rheumatic fever and endocarditis, which are definite enough, conforms closely with that of gout: a result in harmony with probability, and of no small interest and importance.

I am glad that Dr. Longstaff went outside his primary subject of acute rheumatism, and told us something of the recent history of other diseases, particularly of scarlet fever, the prevailing type of which has undergone important changes since the period with which he dealt in an earlier Paper. If statistics of cases were taken, possibly the remarkable inverse relation to rainfall would be found to exist still. Some years ago, I had occasion to examine in some detail the mortality curve of scarlet fever, and came to the conclusion that, apart from any relation to rainfall that might determine the highest or lowest points of the curve, a change in the type of the disease had a great deal to do with the general tendency to rise and fall at intervals of about five years, the years of high death-rate being also those of high case-mortality. In recent years, when the rhythmic curve formerly characteristic of scarlet fever mortality has disappeared, and with it the former parallel curve of case-mortality, the relation to rainfall has disappeared too, and the mortality is persistently low. What all this means I do not profess to know, but one thing is certain, viz., that the relation to rainfall demonstrated in former years was a real one, and I cannot but believe that

in some way or other it is at work still. Whether the death statistics are now too small to bring out this relation, or whether shorter periods, of a month or a week should be compared, or whether the number of days of a certain degree of wetness would afford a better clue than the total amount of rainfall, or whether it is some other condition, such as humidity of air which rainfall connotes, I have little doubt that if the right detail could be followed, we should find that the relation to rainfall exists in one way or another at the present time. And possibly, if we could find a better statistical measure of the quality of scarlet fever than mere case-mortality, something of the old rhythm might still be traced.

On your behalf, and on behalf of the Society, I want to tender our cordial thanks to Dr. Longstaff for his excellent and instructive Paper, which has led to one of the most interesting discussions we have had before this Society. We owe him a great deal for the lessons he has taught us in the past as well as in the present, and for demonstrating the right way to handle mortality statistics so as to get the most good out of them. And I wish to thank him also for putting before us, at very considerable trouble and expense, these admirable diagrams, which are models of clearness.

Dr. Longstaff: Mr. President, Sir William Church, and Gentlemen,—I am very much obliged for the kind way in which you have taken what I had to say to you. There is one thing, at all events, that you have done if you have not exactly encouraged me; you have at least opened to me large avenues of work for the future.

In reference to the powerful criticisms of Sir William Church, it is very difficult to answer them in a few remarks; but my position in this matter is a peculiar one, one which has its happy side and its painful side. Of course, when a distinguished physician gets up and talks about his experiences, a man with none feels that he ought to take a back seat; but there is, perhaps, a point from which medical questions may be approached by those who are outside, without attempting in any way to encroach upon the sphere of the clinician. Out of the large total of certificates given as to the cause of death, naturally there are a large number which cannot be trusted, and allowance must, no doubt, be made for a large margin of error. No doubt a good deal of distrust is felt with regard to these statistics; but that distrust is partly a matter of taste: many people have not the taste to go into them. They have not the

characteristic of liking statistics, and those of us who like them are few. But I think if Sir William Church had spent the same amount of time, and done the same sort of work upon these statistics of the Registrar-General as I have, he would have somewhat more faith in them. The impression produced upon my mind as the result of my investigations is that the collective general practitioner is not such a fool as the public often think he is. And I have come to that conclusion for such reasons as this:—in spite of the natural confusion that may arise between such causes of death as pneumonia, bronchitis, and phthisis, and in spite of apparent carelessness in diagnosis, I nevertheless find that when you take the total results from week to week, or from year to year, that these three different causes of death obey entirely different arithmetical laws. It is quite evident that, in spite of a very large percentage of error, we do find that the figures given for these three great causes of death follow entirely different laws.

When I began my work, my first plan was to plot out causes of death from year to year, and compare them two and two together without any hypothesis at all. I counted to see whether the coincidences were more numerous than the discrepancies. I had made many hundreds of these comparisons, until one night, about twenty-five years ago, just when I was nearly asleep, I was suddenly startled to find that two curves almost absolutely coincided. What, then, were the two causes of death whose curves thus coincided? They were apoplexy and mortification. I then placed these curves beside my meteorological curves, and found that they both agreed very closely with the curve representing cold winters. What is the explanation? In both diseases you have arteries giving way under extra stress: the largely increased blood pressure causes internal hæmorrhage in the one case, while in the other congestion due to cold, coupled with the diseased state of the vessels, causes gangrene. I think this remarkable coincidence is one striking proof that there is a great deal in the Registrar-General's returns.

The difficulty of dealing with rheumatism is, it seems to me, only a fresh incentive to try and get something out of it. It would be impertinence for me to say that I agree with Sir William Church's pathology, but I do entirely agree that great confusion exists with regard to rheumatism. I have, however, tried to show you that when rheumatism is separated into two classes: when the Registrar-General separated from rheumatism, first, rheumatic fever and

rheumatism of the heart, the new group gave a curve which showed the same characteristics as the original curve, but accentuated. Then, when Dr. Ogle comes upon the scene, and transfers acute rheumatism to the said group, I find the curve becomes more defined, and the curve of what is left behind becomes less defined; so that it seems evident that we have got somewhere near to the facts.

What I have called "residual rheumatism" is a hopeless thing to tackle. It includes all that is left behind after you have separated all the elements that you can separate. There is well-founded evidence to show that much of this is really gout. It is well known that these words: "rheumatism," "gout," and "rheumatic gout," are very loosely used. I think, too, it is very likely, as Sir William Church remarks, that senility would account for a great many of these deaths; in that I am entirely at one with him.

As to rheumatism being a zymotic disease, we now know that in the case of ague the proximity of man to man is extremely important, and that although the disease is only found in the neighbourhood of swamps and marshes these are not the cause of the disease; the explanation being that the mosquito is only a feeble flyer, and cannot carry the poison any great distance from one man to another. Some explanation of the same kind may yet be found in the case of rheumatism.

Sir William Church will be glad to know that, some time ago I investigated the statistics of pleurisy, with a view to a comparison with rheumatic fever, and found no resemblance between them.

As regards ulcerative endocarditis, having no personal opinion in the matter, I should agree in the suggestion that it is due to some secondary poison. It appears that public opinion trends in the direction that various infective fevers open the door, so to speak, to a secondary infection, which may be the actual cause of death.

Dr. Newsholme's criticism is much more difficult to meet, because he has gone into statistical inquiries very closely, and I only regret that I have not been able to cover as much ground. I had no intention whatever of implying, by my use of the word "compare," that Dr. Newsholme considers malaria and rheumatic fever to be alike. I do not think there is any grammatical impropriety in "comparing" things which are different. "Contrast" is a result which may come after things have been compared or set side by side.

I should just like to explain what I mean with reference to the progress of our knowledge dealing with rheumatism as it has done with ague. I think, to a certain extent, it has already done so. It used to be considered that malaria was due to dampness, which we now know to be only a secondary and not the prime cause of the disease, and the difference is a very important one.

As to the geographical divisions which I have taken, it is quite true that there are disadvantages in dealing with the whole of England and Wales, but there are also advantages: you get larger numbers, and also you are able, I think, to get certain ideas as to the general prevalence of the disease —in fact, you ought really to deal with it in both ways at once. I think there are more dangers in putting side by side the statistics of different countries for one disease than there are in putting side by side the statistics for various diseases in one country. For example, I remember some time ago, when investigating the statistics of epidemic diarrhœa, some figures were published in reference to the occurrence of that disease in New York. Now, the climatic conditions of New York are so totally different from those of this country that the maximum of the periodic curve occurs at quite a different time of the year, and the comparison is, therefore, apt to be misleading. It is better, for instance, to compare the statistics of rheumatic fever in Christiania with the behaviour of other diseases in Christiania than to compare rheumatic fever in Christiania with rheumatic fever in London.

Someone spoke in reference to this week-by-week variation. My own view of the relationship of this disease to wet or dry conditions is not that it is a question of week to week, but rather of the whole season. In that respect, I quite agree with Dr. Newsholme's measure of ground water. Ground water has been badly treated. I am glad that Dr. Newsholme considers it as a kind of barometer, having no value in itself.

It appears to me that if you look at these curves—take the top curve for rheumatic fever in 1893—this will serve as an answer to what Dr. Bond said about a higher mortality curve in the winter. If you take this epidemic curve, it begins to rise in June, and it goes on rising all through the summer, and attains its maximum in November. Such a disease is not a winter disease, it is rather an autumnal disease, due to some factor which is not immediately removed at the end of the season. We assume that it is due to some infective poison which gets to its maximum

in November, and then begins gradually to die away. Now, if you will compare that with such a disease as bronchitis, which is a winter disease, you will find that the rise occurs very much later, and that the maximum is reached in January or February, during the coldest part of the year. In the case of the twenty years' average also, the maximum for rheumatic fever is in November: it tails off and falls during the cold. It does not rise during the cold.

I should like to call Dr. Bond's attention to the curve at the foot of Diagram C, which shows that gout attains its maximum in January, and diminishes slowly through the spring—that is what I call a winter disease.

I had forgotten the story about the discovery of Salicin. It is curious that it should have been found in places where we now know that rheumatism is not necessarily to be found, and which we do not connect with rheumatism. It seems to be the same with Cinchona. I have not myself seen Cinchona growing in Peru; but in the Nilgiris and on the Himalaya, near Darjiling, I have seen it growing on the most healthy spots, far up the mountain sides, far away from malarial regions.

I am aware that there is some disadvantage in dealing with deaths and not with cases, but still there is something to be learnt from the deaths, and I am simply anxious to show this. I am much gratified to find that my Paper has given rise to so much criticism. I think that does great good.

It would take a very long time even to attempt to reply to the many criticisms, and it would be somewhat difficult, because to a very large extent I agree with those criticisms.

Dr. Nash's contribution to the discussion is most interesting, showing as it does that my figures are in general accord with recent pathological investigations.

In conclusion, I shall be very glad if any work I have done leads to further study of the Registrar-General's certificates in the future by more capable observers.

ON

THE RELATION OF THE PARASITIC PROTOZOA TO EACH OTHER AND TO HUMAN DISEASE.

By E. J. McWEENEY, M.A., M.D., D.P.H., F.R.C.P.I.

(Professor of Pathology, C. U. Medical School, Dublin; Bacteriologist to the Local Government Board for Ireland; Pathologist to the Mater Misericordiae Hospital, Dublin.)

(*Read: Friday, February 17th,* 1905.)

My first duty is to express my sense of the honour conferred on me by the Epidemiological Society in asking me to come forward with a paper on "The Relation of the Protozoa to Disease." This I do most sincerely and heartily. The complexity of the subject, and the energy with which it is being investigated by numerous workers all the world over, make an adequate presentment of our knowledge of it, a task of no little difficulty. In undertaking it, however, there are two circumstances in my favour. In the first place, I selected the "Parasitic Protozoa" as the subject of a Presidential Address delivered about three years ago, when I first took the chair as President of the Pathological Section of the Royal Academy of Medicine (1) in Ireland. In that address I precised, so far as lay in my power, the then state of our knowledge of the subject. This makes it all the easier for me to chronicle on the present occasion the advances that have been made during the three years which have since elapsed. In the second place, the position from which I deal with the subject is not that of an investigator, but rather that of an interested onlooker. The concentration of all the faculties upon some one aspect or department of a subject, necessary as it is for the advancement of science, may in some cases engender a one-sided or distorted view of the relative importance and mutual interrelations of the several branches of that subject. Questions as to priority of new discovery, too, often arise amongst workers, and produce a spirit which is occasionally the reverse of scientific. When a general account of a large subject is required—which I conceive to be the case to-night—the attentive and interested student, with adequate biological training and access to the literature of the subject, is, I believe, in at least as favourable a position for giving it as one who is actually engaged in research. It is from the

standpoint of an onlooker, therefore, that I propose to-night to take a survey of the advances made during the past three years in our knowledge of the disease-producing Protozoa. The outcome of these advances is twofold. They reveal, in the first place, a complexity of reproductive phenomena, quite surprising in such lowly creatures, and constituting a wealth of detail, selection from which is one of the most difficult parts of my task; and, in the second place, an equally surprising parallelism in the developmental cycle of forms which at first sight seem far removed from one another.

It will be remembered that the Protozoa, or One-cell Animals, are divided into four classes: the Rhizopods, the Sporozoa, the Flagellates, and the Infusorians. The Rhizopods are the lowest, comprising mainly Amœbae—shapeless masses of protoplasm. The only one of these which interests us to-night is Amœba Coli.

Many kinds of Amœba occur in the human intestine. Just as the spores of many of the higher fungi, when placed in suitable nutritive conditions, pullulate in the form of yeast-cells, and can only be distinguished from genuine yeast by a study of their development, so it would appear that of the many kinds of Amœbae occuring in the intestine some are *genuine*, viz., the vegetative stage of Rhizopods, whilst others are only Amœboid stages in the development of *higher* forms, such as Trichomonas, Lamblia, and other Infusorians. Genuine Amœbae are divided into those provided with a shell—Thecamœbae, and those that are naked—Gymnamœbae. At least one of the former and two of the latter sort are now known to occur in the intestine. With the representative of the Shell-Amœbae, Chlamydophrys stercorea (Cienkowski), and its complicated life-history,* we need not now concern ourselves, as its habit appears to be purely saprophytic. The two naked forms of Amœbae are genuine parasites. One of them is harmless and the other is one of the most dangerous of pathogenic Protozoa. For reasons based on the laws of zoological nomenclature, the former, which has hitherto been known as *Amœba Coli* (Lösch), has been re-named *Entamœba* by Schaudinn. The second, hitherto called *Amœba dysenteriae* by such authors as admitted its existence (Councilman and Lafleur, Kruse and Pasquale, Koch, Jaeger, and others), has for the same reason been re-named by Schaudinn *Entamœba histolytica.* Schaudinn (2) found out how to distinguish the harmless Amœba from the dangerous one.

* For an account of which see Schaudinn's paper in vol. xix of the *Arbeiten aus dem kaiserl. Gesundheitsamte*, p. 560.

The life-history of these Rhizopods is more complex than was at first suspected. Entamœba Coli occurs very frequently in the fæces of healthy persons, as was first discovered by Grassi. In East Prussia it occurred in half the individuals examined by Schaudinn; of Berliners he found that only 20 per cent. harboured it, whilst at Rovigno, in Istria, it occurred in two-thirds of the persons examined. It is a shapeless mass of protoplasm, not showing a well-marked distinction into clear ecto- and granular endoplasm, and possessed of a large and distinct nucleus. The obstacle to finding it in the healthy fæces is that its *habitat* being in the upper regions of the colon, its vegetative stages die out as the intestinal contents become firmer on their way down. If, however, their downward course be hurried, then the Amœbae can readily be detected. To Schuberg (3) belongs the credit of showing that saline purgatives, by producing liquid motions, enable the Amœbae to be detected in healthy persons. The Amœbae have two distinct cycles of development: one vegetative or asexual, taking place in the naked state, the other displaying a primitive but unmistakeable form of sexuality, and occurring inside of a capsule resembling an egg-shell and termed a cyst. In the vegetative form, the Amœbae either simply split in two, or their nucleus divides into eight daughter-nuclei, each of which takes a portion of the protoplasm so as to form a characteristic brood of eight young Amœbae, which come apart. In the other, or sexual cycle, the Amœba rounds itself off, comes to rest, contracts, and surrounds itself with a gelatinous coat which becomes the cyst-wall. The two nuclei, after undergoing reconstruction and chromosomic reduction, divide into halves, which copulate, so as to form two fresh nuclei, each containing half of the two parent-nuclei. Each of these copulation-nuclei now divides twice, and the divisions form eight young Amœbae, which, however, cannot leave their cyst until it has been taken in by a new host, and has had its wall softened in the stomach and duodenum. The resemblance in general outline to the developmental process of the Sporozoa will be at once manifest: asexual swarming in the primary host till it is exhausted or conditions become unfavourable; then sexuality with cyst-formation and change of host. The details are different; the underlying idea, so to speak, is the same.

Let us now take a glance at the pathogenic rhizopod Entamœba histolytica. Its vegetative stage differs from that of the non-pathogenic Entamoeba Coli in, firstly,

possessing a clear, tough, outside layer or ectoplasm which enables it to penetrate between the epithelial cells, and, secondly, in not possessing a distinct, well-defined nucleus. Its tough ectoplasm enables it to force its way into the deeper layers of the mucous membrane, where it multiplies and forms the undermined ulcers which are so characteristic of tropical dysentery, and are readily distinguished from the diphtheritic appearance presented by the bacillary form of the disease. Schaudinn (4) has actually observed these Amœbæ in scrapings of the freshly-excised bowel of the experimentally-infected cat, crawling around for hours and pushing in between the epithelial cells. The two sorts of Amœbæ differ also in their reproduction. The pathogenic form, in its vegetative stage, divides into two, or forms new individuals by budding. Brood-formation does not occur. The process of encystment is also quite different. It comes on when the patient is beginning to recover from his attack of dysentery and the fæces are becoming solid. The nucleus gives up most of its chromatin in granular form to the plasma, and its remains are expelled. The plasma now projects from its surface a number of little knobs, each containing a particle of chromatin, and measuring from three to seven micra in diameter. These break off after a while, and each becomes surrounded by a capsule which ultimately becomes quite brown, hard, and opaque. These "spores" are then expelled with the fæces, and serve to infect a fresh host. It will be seen that the indications of a sexual process are much less distinct in this species, owing perhaps to the difficulty of ascertaining precisely what goes on during the maturation of the minute spores.

In my Address, already referred to, I spoke of the *rôle* of Amœbae in the following terms:—"The general conclusion to which I incline, after a careful survey of the literature, is that the Amœba Coli is, in all probability, either wholly or partially responsible for the causation of one form of dysentery—that which is termed 'tropical,' and is not endemic in this country." Subsequent investigation has borne out this view. One of Schaudinn's experiments seems quite conclusive. He allowed a small fragment of fæces from a case of dysentery, contracted in China, to dry in the air, suspended it in water, made a number of slide and cover-glass preparations, and by searching them systematically throughout on the mechanical stage, he satisfied himself that they contained nothing resembling the cysts of *Entamœba Coli*, but only the small brown spores of *E. his-*

tolytica. Of course there were no free Amœbæ, as these had been destroyed by drying. The cover-glasses were then removed and the fæces washed off with clean water, made up to about 1 cc., and administered with meat and milk to a young, healthy cat, the fæces of which had been proved by careful examination to be free from Amœbæ and their cysts. Three days afterwards, the cat began to pass slimy fæces, streaked with blood. These were found to be swarming with the typical *Entamœba histolytica.* Next day the animal died of dysentery. The autopsy showed characteristic ulceration of the large intestine, with crowds of Amœbæ in all stages of penetration into the intestinal wall. There were, of course, no cysts, the animal having died in the acute stage of the disease. In order to see whether the Amœboid stage could propagate the disease, Schaudinn administered large quantities of this fæcal matter to another cat. It remained quite healthy. Its fæces were examined for four weeks, and no Amœbæ were detected. It was then given a small portion of the same dried-up cyst-containing fæces from the Chinese case that been used in the other experiment. Within six days, Amœbæ appeared in the fæces. This cat, which was older and stronger than the previous one, developed dysentery, and died of it in about a fortnight. From this it would seem that the Amœba-stage introduced by the mou this not capable of producing infection. The older experimenters, from Kruse and Pasquale onwards, had often succeeded in producing infection by introduction of Amœbæ *per rectum:* and Jürgens (5) has again successfully performed the experiment. This rectal transference can, however, hardly be realised under natural conditions; and, accordingly, it is to the dried-up cyst-containing fæces present in dust and water that we must look for the propagation of dysentery. Whether the Amœbæ contained in the fæces from the acute stage will, if gradually dried, become converted into cysts *outside the body,* or whether the Amœbæ that are going on to cyst-formation receive their impetus along that track whilst still in the bowel, does not seem to be quite clear. In any case, the practical importance of these observations is obvious. The cyst-containing fæces of cases of dysentery must be prevented from getting access to drinking-water, or from being conveyed by flies and deposited on food. There would seem, by the way, to be a sort of mutual exclusiveness between *Entamœba histolytica* and the Shiga dysentery-bacillus. Schaudinn failed to find them in the same intestine, and Castellani (6) has recently had the same experience in Ceylon.

The next class is that of the Sporozoa, so called because at one stage of their life-history the individual breaks up into a number of minute reproductive bodies resembling the spores of fungi or mosses, and, like them, often contained within a cyst or thick-walled capsule. Amongst the chief orders of this Class are the *Gregarines*, which mostly live in the intestine of crustaceans and insects; the *Coccidia*, many of which inhabit the digestive tract and its annexes in mammals, birds, and various invertebrates (myriapods, etc.); and the *Hæmosporidia*, or parasites of the blood-corpuscles, which are the cause of malaria, and to which, therefore, a very special interest attaches. All these organisms, however different their vegetative stages, have in common a certain ground-plan of life-history, varying in individual cases, and with regard to matters of detail, but the general features of which pervade the whole Class. This life-history comprises two main features: alternation of generations and change of host. Alternation of generations means that a sexual method of reproduction alternates with an asexual method; whilst the term "change of host" does not, of necessity, imply a change of host-*species*, but in many cases only a change of host-*individual*. The general idea underlying the parasitism of these organisms is that, once the sexually-produced reproductive body, or spore, finds its way into a suitable host, and into a suitable cell of that host—for they are *intra*-cellular parasites—it rapidly attains its full size, and splits up, without the intervention of sexuality, into a number of fragments which fall asunder, and, invading fresh cells of the same host, repeat the process. By this means, the host-animal becomes speedily pervaded by swarms of parasites, and the resultant disturbance of function is the disease. After a time, owing to the production of specific anti-bodies, or the exhaustion of the reproductive power of the parasite under the strain of repeated asexual division, this process gradually ceases, and specially-differentiated male and female forms appear and copulate. The fertilised female forms a cyst containing spores, which have to be transmitted to a fresh host before they can develop. The points at which the greatest variability obtains are, firstly, the place and time of conjugation, and secondly, the arrangements for transfer from one host to another. Where the organism passes its asexual stage in the blood, transfer must necessarily be effected by the only agency whereby blood is at all frequently removed from the vessels, *i.e.*, blood-sucking invertebrates, such as flies and ticks. The intermediate

host of blood-inhabiting parasites is, therefore, an invertebrate blood-sucker. Some of the blood-parasites—those of malaria, for example—avail themselves of the invertebrate host as the scene of their sexual reproduction, and multiply enormously within it. Others, such as the Trypanosomes of the Mammalia, remain, so far as we are at present aware, quite passive within their cold-blooded host, and use it merely as a vehicle.* Others, again (some of the Piroplasmata), in some mysterious way pass on into the eggs of the invertebrate host, and infect its young brood. On the other hand, when the parasite passes its asexual stage in a cavity of the host that is connected with the outer world (the Coccidia), the problem how to escape is comparatively simple, and an intermediate host may be dispensed with.

In order to readily characterise these several stages, and to emphasise homologies, it is necessary to adopt a systematic nomenclature, and I accordingly use that elaborated by Schaudinn and Lühe, according to which

The name Schizont is applied to the mature asexual individual.

" Merozöites is applied to the pieces into which the Schizont splits up.

" Gametocytes is applied to the forms which produce or become converted into the sexual individuals, viz. :—

Macrogametocyte	The *asexually-produced* individual which, by chromosomic reduction, becomes converted into a female, and
Microgametocyte	The *asexually-produced* individual, which, by chromosomic reduction and nuclear division, becomes converted into a *number* of males.
Gametes	The sexual individuals, viz. :
Macrogametes	Females (the ova of the Metazoa), and
Microgametes	Males (the Spermatozoa of the Metazoa, Antherozoids of the higher Cryptogams).
Zygote	The fertilised Macrogamete.
Öokinet	The Zygote when capable of locomotion.
Öocyst	The Zygote when it has come to rest, and is surrounded by a thick capsule.
Sporoblasts...	Secondary cysts or spore-bags formed within the öocyst.
Sporozöites	The final products of the act of sexual reproduction—minute bodies formed often in large numbers within the öocyst, and which, on being removed to the new host give rise to the schizonts.

* Since this was written, Schaudinn's former assistant, Prowazek, claims to have observed (*Arb. a. d. kaiserl. Gesundheitsamte*, 1905, p. 376) sexual forms of the rat-Trypanosome (*T. Lewisii*), in its intermediate host the rat-louse (*Hæmatopinus*).

I come now to what has been done on the parasites of human malaria during the last three years. The details of their life-history as established by the labours of Laveran, who discovered them; Golgi, who differentiated them into species; Ross and Manson, who discovered the change of host; MacCallum, who recognised their sexuality; and Grassi, who identified the mosquito, have been more fully worked out, and numerous important details have been added. *Plasmodium vivax*, the cause of tertian ague, has been minutely studied by Schaudinn (7) at an ague-stricken village near Rovigno on the Adriatic, where he had abundant material. Some of his observations regarding the sexual process are of special interest. It starts in from ten minutes to half an hour after the blood has been sucked in by the gnat. The microgametocyte, which is readily recognised by its hyaline plasma, large oval nucleus, and coarsely granular pigment, suddenly shoots forth from four to eight curved filaments, 20 to 25 micra long and 1 micron at thickest. Meanwhile the macrogamete has been preparing itself for fertilisation by ejecting part of its nucleus. This process appears to be homologous with the extrusion of the polar bodies by the Metazoan Ovum. The number of chromosomes is thus reduced by one-half. The extruded matter may possibly, by its disintegration, provide a diffusable substance acting chemotactically on the microgametes and causing them to approach the female, in the same way as the canal-cells of the archegonium of ferns yield malic acid which attracts the Antherozoid. The female then protrudes a "receptive eminence," and as soon as one of the males has adhered to this, it is at once withdrawn carrying the struggling microgamete in with it. In the case of the malarial parasites only the one male obtains admission, but in the Coccidian *Cyclospora caryolytica*, which causes an infective form of enteritis in the Mole (Talpa), what is called *polyspermy* occurs (8). Quite a number of male gametes force an entrance into the macrogamete. Only one of them undergoes the fertilising fusion, whilst the others break down and serve as food for the developing zygote. The presence of superfluous males, however, is apt to disturb the proper relation of male and female pronucleus, and give rise to interesting pathological changes described by Schaudinn. In the Tertian parasite only one favoured microgamete gains access, and no sooner has it done so, than a coat of mucus is secreted by the female, which keeps away the excluded males and

is homologous with the cyst-wall of the Coccidia. The zygote now becomes a motile öokinet, and this becomes an öocyst, which comes to project, as is well known, into the body cavity of the mosquito, and the internal plasma of which splits up into sporoblasts, which in turn split up into sporozöites. Some of the liberated sporozöites, according to Schaudinn, obtain access to the ovary of the gnat and infect the developing ova, so that the young brood next spring harbours the parasite, and the young mosquitoes can confer the disease without having previously sucked blood—a fact of great epidemiological importance, if it be confirmed by subsequent observation. Finally, Schaudinn succeeded in observing the entrance of the sporozöite into his own red corpuscles, and thus completed the developmental cycle.

I now come to the last part of my subject, namely, the Order of Flagellates, to which belong the Trypanosomes. Three years ago, in the Address already referred to, I gave some account of these parasites in connection with the diseases of animals known as Nagana, due to T. Brucei, and Surra, due to T. Evansi, and I described the *rôle* of the Tsetse fly as carrier of the latter. I also referred to my own experience of these parasites, gained on the only species accessible to me at the time—the Trypanosome of the common sewer-rat, T. Lewisii. During the three years that have since elapsed, the *rôle* of these parasites as disease-producers amongst the lower animals has been greatly enlarged. Dutton and Todd (9) found a disease of horses in Gambia to be due to T. dimorphon. Elmassian, (10) followed by Voges and Lignières, have shown that the disease of horses in South America known as Mal de Caderas, is due to another Trypanosome (T. equinum). The sexual malady of stallions and brood-mares known to the French as Dourine, or Mal de Coït, is due to T. equiperdum, which was first discovered by Rouget in 1896 (11). The so-called Galziekte or Gall-sickness of Bovines in the Transvaal has been shown by Theiler to be caused by a species called after him T. Theileri (12). Other forms have been described in the smaller Mammals, such as, the rabbit, guinea-pig, dormouse, bat, as well as in birds, reptiles, amphibians, and fishes. But the great access of interest in the study of Trypanosomes is due to the fact that they are now known to produce disease in the human race. In December, 1901, Dutton (13) was the first to recognise as a Trypanosome the *vermicule* previously seen by Forde in the blood of a European

patient in the Hospital at Bathurst. He described it accurately, and named it T. Gambiense. In April, 1903, Castellani (14) discovered a Trypanosome in the cerebro-spinal fluid of natives of Uganda affected with sleeping-sickness, and this malady has consequently come to be regarded as one of the manifestations of human Trypanosomiasis. David Bruce (15) with his fellow-workers Nabarro and Greig, brought home the propagation of the disease in Gambia to the fly *Glossina palpalis*. Finally, there is the discovery, so far back as 1900, by Leishman, and, independently in 1903, by Donovan, in smears of spleen-pulp from cases of splenic cachexia in India, of parasites which Rogers (16) has shown to be stages in the development of a Trypanosome. These, shortly summed up, seem to me the most important landmarks in the development of our knowledge as regards Trypanosomes.

The side of the question which I propose to deal with this evening is the relation of Trypanosomes to other Protozoa, more especially to the malarial parasites, and to certain organisms known as Spirochætæ, and hitherto regarded as Bacteria.

A correct idea of the structure of a Trypanosome is essential to an understanding of what follows. Its fish-like body, twice or thrice as long as a red corpuscle, is fringed along one side with the undulating membrane, which is prolonged at the end now generally looked upon as anterior, into the flagellum. In the body is a large nucleus, and there is also, towards the posterior end, a smaller dot-like mass of chromatin close to which originates the margin of the undulating membrane—the edge which is prolonged into the flagellum. The nature and homologies of this smaller mass of nuclear substance have given rise to lively discussion. Some term it a centrosome, others a micro-nucleus. There seems to be no doubt that it gives origin to and presides over the locomotive apparatus—that the edge of the undulating membrane and the flagellum (both of which give the colour-reaction of nuclear substance by the Romanowsky method) are part and parcel of this mass, which I shall therefore term the *blepharoplast* (literally, flagellum-former). Trypanosomes bear a close resemblance to the spermatozoa of some animals (Bombinator), and are constructed on similar lines. They multiply mostly by unequal longitudinal division, more rarely by multiple division (rosette-formation). The first part to divide is always the blepharoplast. They are agglutinable by foreign or immune serum, as was shown by

Laveran and Mesnil. To Professor F. G. Novy, of the University of Michigan (U.S.A.), and his collaborator, Dr. W. J. MacNeal, belongs the great merit of discovering a culture-method applicable to Trypanosomes (17). By its means they have succeeded in growing the Trypanosomes of the rat, of Nagana and of Surra, on an artificial medium (blood-agar), and have recently cultivated several forms from the blood of birds. Until the researches of Schaudinn, which I am now about to detail, Trypanosomes were looked upon as independent organisms, and were not known to possess any trace of a sexual mode of reproduction. It appears to me that the discoveries of this brilliant and industrious worker have not as yet been received in this country the attention to which they are entitled.

In the blood of the barn-owl (*Athene noctua*), Schaudinn (18) found three distinct protozoal parasites. First there was the Proteosoma, the development of which in the mosquito, *Culex nemorosus*, had been so brilliantly discovered by Ross and subsequently studied by Koch. Then there was another plasmodial form of parasite of the red corpuscles *Halteridium*, previously studied by MacCallum and Koch; and finally there was the so-called *Leucocytozöon*, discovered so far back as 1894, by Danilewsky. The two last-named parasites own *Culex pipiens* as their intermediate host.

Previous observers had followed the development of *Halteridium* (called by Schaudinn *Trypanosoma noctuæ*) as far as it goes on in the hanging-drop of blood. Further they could not pursue it, not knowing the species of mosquito which serves as its intermediate host. Schaudinn having ascertained that it is propagated by *Culex pipiens*, found that, as the result of fertilisation, an öokinet is formed which moves along like a slug by the aid of its own mucus towards the front of the mosquito's stomach and there becomes converted into a Trypanosome. Omitting the minuter details, the process is as follows (Fig. 1). The nucleus divides unequally, so as to form a hetero-polar spindle. The larger (left) half enters the resting stage, whilst the smaller (right) half, which is now in the ectoplasm at the periphery, again divides, forming another hetero-polar spindle, with the long axis parallel with that of the öokinet and the smaller pole forwards. The chromatin of this spindle forms the skeleton of the locomotive apparatus of the future Trypanosome, the thickest thread forming the free edge of the membrane, whilst the posterior centro-some becomes the blepharoplast. Thus a Trypanosome is formed, and of these Schaudinn describes three types: the "indifferent," the "female," and the "male."

The öokinet, which is to develop into a "female" Trypanosome, has darker protoplasm and a smaller nucleus than the form which he calls "indifferent." It goes through the same nuclear processes, the result being the formation of a larger Trypanosome, which is provided with a relatively small flagellum, and is, therefore, less active in its movements. These become limited to slight bending and twisting. The "female" Trypanosome ultimately comes to resemble a mature Gregarine, and possesses a marked power of resisting external unfavourable influences. If, for instance, the mosquito is starved and then kept on ice, all the parasitic forms within it die out save the "female" Trypanosomes, which lie buried between the epithelial

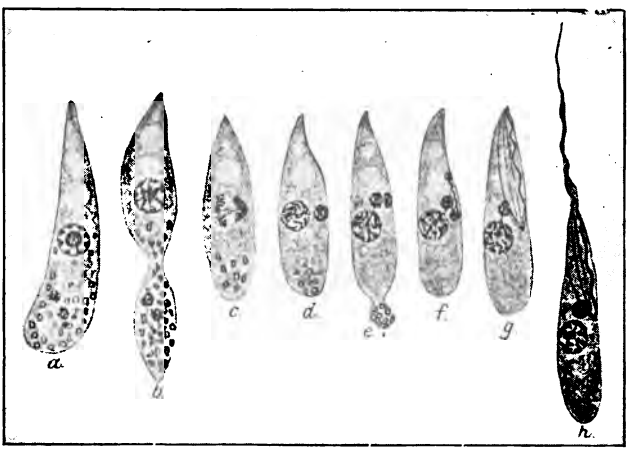


Fig. 1.—*Trypanosoma (Halteridium) noctuæ* (Schaudinn).

Stages in the development of the Öokinet into the "indifferent" type of Trypanosome (after Schaudinn): *a*, the slug-like öokinet, showing its complex conjugation-nucleus (synkaryon), near the middle, whilst in its lower part are seen the remains of the reduction-nuclei, and pigment-granules, the protoplasm containing which is being removed in *b* by abstriction; *c* and *d*, the first unequal (heteropolar) nuclear division; *e*, the second ditto; *f*, the third ditto, and first appearance of edge of undulating membrane; *g*, *h*, further development of undulating membrane and flagellum. (All highly magnified.)

and elastico-muscular coat of the intestine, last through the winter, and infect the brood of eggs laid next spring. Multiplication of this Gregarinoid stage takes place by a process termed by Schaudinn "parthenogenesis," and which he homologises with the re-infection of the warm-blooded host in Malaria, the so-called "relapses." The forms which persist in the blood of old-standing cases of malaria are,

according to him, old females (macrogametes) [? macrogametocytes]. After a time these "sink" to the level of schizonts; in other words, produce a brood without having been fertilised. This mode of reproduction—the skipping of the sexual generation—appears to me to be homologous with the phenomenon amongst ferns, first described by De Bary under the name of "apogamy."

The öokinet which is to form the "male" Trypanosomes, is distinguished *ab initio* by its small size, large nucleus and absence of reserve material in its protoplasm (Fig. 2.) By a process of reduction it gets rid of one half of its nucleus—the half which is preserved in the female—and the remainder splits up so as to form eight minute Trypanosomes. These he regards as microgametes which, however, are unable to perform their physiological function of fertilisation. This act is only performed at one period of the life-

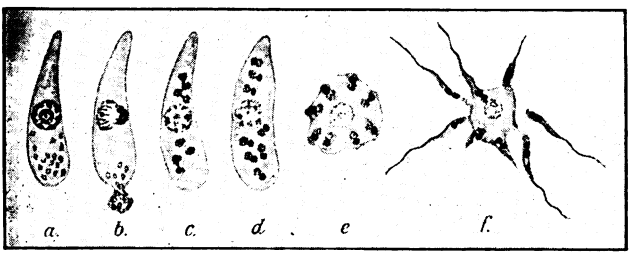


Fig. 2.—*Trypanosoma* (*Halteridium*) *noctuæ*.

Development of "male" Trypanosomes from the Öokinet (after Schaudinn); *c*, *d*, gradual disappearance of larger half of nucleus, whilst from the smaller half the microgametes are developed (*e* and *f*).

history of the parasite, viz., within a few minutes of its arrival within the stomach of the mosquito.

As the result of these processes, the intestinal canal of the mosquito becomes inundated with "Trypanosomes" of all three kinds. Sometimes they are produced in such swarms that their resting-stages occupy all the epithelium, and the mosquito dies of the disease. Ordinarily, the insect feeds three times, and it is during these three periods of digestion that the parasites make their way down the bowel, through the wall of the colon, and so into the blood-stream, whereby some get carried to the ovaries, where they infect the young brood; most, however, are carried to the front of the body-cavity into the head, where they accumulate round the pharynx, and ultimately rupture into it, and so obtain access to the lumen of the proboscis. The

mechanism of transference to the warm-blooded host is extremely interesting, and as the parasites of human malaria may possibly be transferred in the same way, it seems worth while to mention the details. *Culex pipiens* possesses œsophageal diverticula which become distended with gas, and may therefore be termed, for brevity's sake, "gas-bags." The gas is carbon dioxide, which is evolved by a sort of yeast-like fungus always present in the insect's stomach, from traces of glucose present in blood, or—much more abundantly—from glucose present in the plant-juices which the insect occasionally sucks. At the commencement of the act of suction, when the insect has its proboscis buried in the skin of its victim, its body undergoes one or more violent contractions, which eject the contents of its fore-gut and gas-bags into the skin. These contents comprise gas, saliva, and whatever particulate matter is present, viz., yeast-cells and sporozöites. Schaudinn looks upon this contraction as a sort of dyspnœal effort due to the entrance of the CO_2-laden air, which immediately bathes the skin, into the tracheæ of the insect. He succeeded in producing the contraction artificially by placing an infected *Culex* on a cupped-slide, with its proboscis in a drop of glycerine under a cover-glass, and its body projecting over the hollow. In this he evolved some CO_2 from a fragment of chalk and a droplet of acid; and he observed that the gnat's body underwent a violent contraction, which had the effect of expelling into the glycerine its contained gas, yeast-cells, and sporozöites. Viewed teleologically, the effect of the CO_2 would be to paralyse the thrombocytes, and in other ways to delay the coagulation of the blood. The hyperæmia and pain caused by the bite he considers to be due to the enzyme of the yeast-cells. He dissected out the "gas-bags," and pushed them into a fine puncture in his own skin, with the result that the typical swelling, redness, and itching came on at once. The salivary glands he found quite inoperative in this respect. The irritating effect of the "gas-bags" appears to depend on the quantity of yeast-cells contained in them, for it was much more marked when the yeast had been allowed to proliferate actively as the result of feeding the gnat on plant juice. The fungus is not a true yeast, but a yeast-like stage in the life-history of one of the *Entomophthoreæ*—fungi with which we are all acquainted in the shape of the well-known *Empusa muscae*, which kills flies in the fall of the year, and causes their bodies to adhere to the window-pane, surrounded by a white cloud of ejected conidia.

Let us now follow the Trypanosomes into the blood of the owl, whither they are carried by the bite of the *Culex*. Such "males" as are conveyed in the fully-differentiated state die off. The mature "females" are usually too large to pass the gnat's proboscis. It is the "indifferent" forms and young "females" that thrive in their new surroundings. Adhering by their flagellum to the red corpuscles, they bore their way in, withdraw their flagellum, and grow larger in the plasmodial form described by MacCallum, Koch, and other observers. They do *not* proceed to form asexual rosettes (schizonts), a fact expressly drawn attention to by Koch, who could not find in the blood of his artificially-infected sparrows and pigeons any trace of this form of multiplication, which is so abundantly met with in

Fig. 3.—*Trypanosoma* (*Halteridium*) *noctuæ*.

a, attachment of "indifferent" Trypanosome to a blood-corpuscle of the owl, by its flagellum. Another individual, *b*, has completely penetrated into the corpuscle and has lost its flagellum; *c*, increase in size, and *d*, escape of the intra-corpuscular parasite; *e*, *f*, *g*, further stages of growth. (After Schaudinn.)

human malaria. What actually happens is this. The intra-corpuscular parasites leave their host-cells only during the night, and in the deepest recesses of the circulation—the spleen and bone-marrow. There they swim about for a while as free Trypanosomes (of all three varieties above described) (Fig. 3). During the day they mostly go back to the corpuscles. Thus they change about at intervals, for six days, by the end of which time they have attained their full size. Finally emerging, the full-grown parasite now multiplies by repeated acts of longitudinal fission till the products have attained their minimal size. The excessively minute flagellates so formed penetrate into fresh red

corpuscles, till the blood become fairly inundated. Then they die down, and only a small number of "indifferent" forms survive, which become gradually evolved into macro- and micro-gametocytes, and can only complete their evolution in the body of the gnat. In concluding the *resumé* of the remarkable life-history of Trypanosoma (*Halteridium*) noctuæ, as observed by Schaudinn, I would point that of previous observers only Danilewsky seems to have come across the Trypanosome. Koch, in his careful study of the parasite, does not seem to have observed this stage; at any rate, he makes no mention of having seen Trypanosomes in the blood of his birds infected with *Halteridium*.

I have dwelt thus at length upon the history of this parasite on account of the light it throws upon the nature of the Leishman-Donovan body. These parasitic bodies are contained in large endothelial cells of the spleen and marrow: so, for a time, are the *Halteridium*-parasites The Leishman-Donovan bodies possess two nuclei—a large oval and a small rod-shaped one,; so do the involuted intra-corpuscular Trypanosomes of *Halteridium*. Finally, we have the brilliant discovery, by Rogers, that blood containing Leishman-Donovan bodies gives rise to Trypanosomes. The details given by Rogers tend to support the supposition that the life-history of the parasite of cachexial fever runs on the same lines as those of owl-malaria. For example: "the splitting-up of the parasite into two, the separation usually beginning at one end," suggests the longitudinal division of the Trypanosomes; and "the presence of smaller pear-shaped flagellated bodies like those described by Plimmer in Tsetse fly disease," which are evidently involution, or immature forms, also described by Laveran and Mesnil in their recently-published text-book (19), under the name of *mises en boule*, and figured on p. 156. Finally, there is the fact that the Trypanosomes were only developed in Major Rogers' cultures at a low temperature (22 deg.), which would point to their being naturally produced outside the warm-blooded host in a blood-sucking Invertebrate, such as a bug or mosquito. There can be little doubt that the original opinion of Major Leishman with regard to these bodies is correct, and that Kala-azar and cachexial fever will prove to be a form of human Trypanosomiasis. The same opinion doubtless holds good of the bodies found in the granulation tissue of Delhi-boil and Tropical ulcer, by Cunningham, Wright, James, Christophers and others.

Remarkable as is the connection so established between

the malarial parasite and the Trypanosome, it is, at any rate, a connection between one Protozöon and another. But the developmental cycle which I am now about to recount will, if established by further investigation, effect a still more surprising connection, viz., between a malarial parasite and an organism hitherto looked upon as a Bacterium, Spirochæte.

The malarial parasite in question was discovered so far back as 1891, by Danilewsky, of Charkow (20), in the blood of *Athene noctua*, and called by him the Leucocytozöon, from its dwelling in the white corpuscles. It was afterwards seen and depicted by Ziemann, (21) in his well-known work on blood-parasites. Both its warm-blooded and cold-blooded host (*Culex pipiens*), as well as its general aspect, are identical with those of *Halteridium*. In the mosquito's

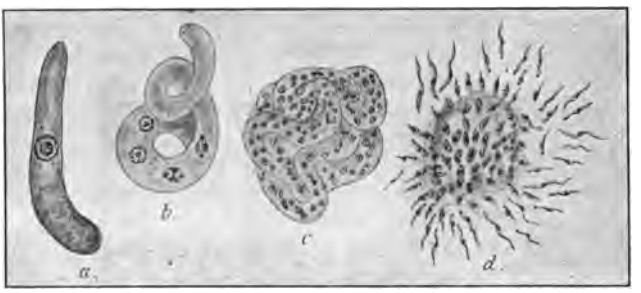


Fig. 4.—*Spirochæte Ziemanni*. (After Schaudinn.)
Development of minute Trypanosomes (*Spirochætæ*) from Öokinet (see text).

stomach its stationary macrogametes are fertilised by the motile microgametes. An öokinet (*a*) is thus formed, which becomes spirally coiled, so as to form a sort of corkscrew (*b*), the coils of which coalesce (*c*) (Fig. 4). To my mind, this process is remarkably like what occurs in the development of the sexual fructification of the cleistocarpous Ascomycetes, such as Eurotium. The original nucleus divides into a vast number, each of which presides over a territory of protoplasm, which gradually becomes differentiated into a Trypanosome-like body, with blepharoplast, undulating membrane, and flagellum (Fig. 4, *c*, *d*). Of these, as in *Halteridium*, there are three sorts—"indifferent," "female," and "male"—and all are very minute. As the result of repeated division, the individuals may become so small as to become not only incapable of measurement, but even of being perceived individually with the highest magnifications. It is

only when agglutinated in rosette form that they can be well seen; and Schaudinn, who describes these remarkable appearances, has no doubt that they would constitute a *filtrable* virus: that is, one capable of passing the pores of a porcelain filter, and therefore in the same category as those of pleuro-pneumonia, foot-and-mouth disease, and perhaps of small-pox. These minute Trypanosomes increase greatly in length, and become spirally twisted round their long axis, so as to constitute what have hitherto been termed Spirochætes. Now the leading pathogenic Spirochæte hitherto known is S. Obermeieri, the cause of Relapsing Fever. It has been observed to move equally well in either direction, "forwards" or "backwards." But Trypanosomes always move forwards, *i.e.*, flagellum first, and this would seem to be a serious difference between them and Spirochæte.* Schaudinn states, however, that these long, thin Trypanosomes divide longitudinally, and that the two new individuals *remain coherent by their hinder ends*, and can accordingly move in *either* direction. They continue to cohere until each individual has again divided in the same way. These Spirochæte-Trypanosomes agree with the Trypanosomes of the Mammalia, and differ from those of birds (*Halteridium*), in becoming agglutinated by their *hinder* ends. It is at the end of the first digestive period of the mosquito that these Spirochæte-like Trypanosomes are developed from the ookinet, inundate the body-cavity, and infect the ova. On re-introduction, natural or experimental, into the owl, they begin by multiplying enormously in the Spirochæte form before proceeding to the formation of sexual individuals. It is surprising to me that neither Danilewsky nor the other workers who observed the intra-corpuscular stage of this parasite should have seen the Spirochæte. Possibly, their birds were old cases, in which all the parasites had passed on into the intra-corpuscular resting stage. The cell preferred by this parasite is not the mature red corpuscle, nor yet the mature leucocyte, but the hæmoglobin-free precursor of the former—the erythroblast. This accounts for its presence mainly in the spleen and marrow (*cf.* the behaviour of the Leishman-Donovan body). In human relapsing fever, Metschnikoff observed that when the Spirochæte disappears from the blood at crisis, it accumulates in the spleen. It would be interesting to know whether Metschnikoff observed any flagellated or plasmodial forms amongst the spleen-cells. If the development of human

* Prowazek (*loc. cit.*) now holds that this is not always the case.

relapsing fever really proceeds on the same lines as *Spirochæte Ziemanni* of the owl, so accurate an observer as Metschnikoff could hardly have avoided seeing them, more especially as Schaudinn describes the sexual forms as relatively very large: so large as to be unable to find entrance into their host-cell, but rather drawing it into themselves, and provided with a distinct undulating membrane. Ziemann also in his figures misses the flagellated form, though his figure of the peculiar way in which the erythroblast-nucleus is flattened out exactly agrees with that of Schaudinn. The parasites undergo successive stages of intra- and extra-corpuscular existence in the blood

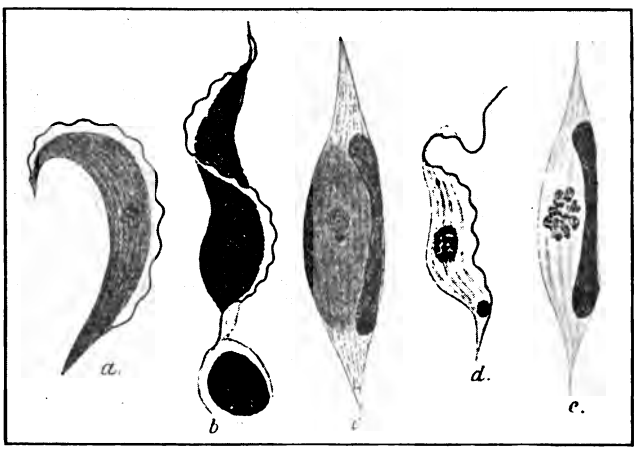


Fig. 5.—Trypanosome-like Stages of Spirochæte Ziemanni in the Blood of the Owl (after Schaudinn).

a, motile stage of full-grown female (the female forms possess the undulating membrane, but not the flagellum); *b*, the same, showing spiral twisting during motion, and an erythroblast of the owl adherent to the *hinder* end of the parasite; *c*, resting stage with transparent outer sheath (periplast), the dark structure to the right is the nucleus of a digested erythroblast; *d*, active stage of the microgametocyte; *e*, resting stage of same, showing its nucleus already divided up, and the nucleus of a digested erythroblast to the right.

of the owl (Fig. 5), and on removal from it by *Culex pipiens*, at once proceed to the formation of large rounded macrogametes with dense protoplasm, and small active microgametes which look like a minute Trypanosome or a Spirochæte. Fertilisation gives rise to the öokinet from which we started.

Should this developmental cycle be confirmed by other observers, then it follows that Trypanosoma and Spiro-

chæte are really only different names for the same thing. The following features about relapsing fever would then be accounted for: the non-cultivability of the Spirochæte on ordinary substrata, and the persistence of the parasite in the spleen after its disappearance from the general circulation. It would also appear probable that its propagation would be effected by some blood-sucking parasite. This has actually been found to be the case with the "Spirillosis of fowls," recently described by Marchoux and Salimbeni (22). They found that the Spirilla were conveyed by the bird-tick, *Argas miniatus*. Unfortunately, they give no details as to what happens to the parasite inside of the tick; and although they describe the spleen as enlarged to three times its size, they give no account of its minute appearances.

I am not aware that the intermediate host of the Spirillosis of ducks described by Sakharoff, nor of that of oxen quite recently described by Laveran (23), has been ascertained.

In the case of human relapsing fever, it seems most probable that bed-bugs carry the infection. Karlinski (24) found abundant Spirilla in the bugs captured in Bosnian houses where the inmates suffered from the fever, whilst he could find none in bugs obtained from houses where there was no fever. He found that the Spirilla persisted for 20 days in the bugs. Marchoux and Salimbeni say that their Spirilla persist in *Argas* for 100 days.

As I pen these lines, another Spirillosis of the human subject has been discovered. Major Ross, F.R.S., announces in the current number of the *British Medical Journal* (Feb. 4th, 1903) that human "tick" fever in the Congo has been found by Dutton and Todd to be caused by Spirilla which are conveyed by the tick *Ornithodorus*.

The question is, of course, still open whether the Spirilla of these diseases are really built on the same lines as the Spirochæte Ziemanni of Schaudinn. So much would thus be explained that it is certainly a very tempting hypothesis.*

This Paper, already far too long, would be swollen to inordinate dimensions were I to attempt to refer in detail to the *Piroplasma* parasites. I am, however, absolved

* As I correct these lines for the Press, the interesting announcement has just reached me of the discovery by Schaudinn, in various syphilitic lesions, of extremely delicate, actively motile Spirochætæ, which he is inclined to look upon as the cause of the disease. The paper appears in the *Arb. a. d. k. Gesundheitsamte*, Bd. xxii, p. 527, and is dated April 10th, 1905. I have since confirmed this discovery.

from the task by the fact that this Society has already been this season the recipient of a very admirable communication from Prof. G. H. F. Nuttall, F.R.S., on the subject of Ticks and Tick-transmitted Diseases, which practically covers the ground. I will merely mention that Kossel and Weber (25), of the German Imperial Health Office, have found Trypanosome-like stages in the blood of cows affected with haemoglobinuric fever, and also in the intestines of ticks that had sucked the blood of such cows. Furthermore, Theiler, and quite recently Bowhill (26), have found both Trypanosome and Spirochæte forms in the blood of oxen and dogs affected with Piroplasmosis.

Judging by analogy, it seems not improbable that the hitherto unseen parasite of Yellow Fever may prove to be a Spirochæte too small to be seen with the highest powers of the microscope.

[Dr. Dutton has since succeeded in conveying spirillum-fever to the monkey by means of the tick, and further, in proving that young ticks reared from naturally-infected parents can convey the disease at their first feed—an observation of the very highest significance, in view of the fact that the tick fever or infectious hæmoglobinuria of cattle ("bovine malaria," as it is sometimes called) is propagated, not by the naturally-infected tick, but by its progeny. That Dutton's brilliant career, already so rich in achievement, should have been brought to an untimely end by infection received during his investigations, is an announcement which will cause profound regret, more especially to those engaged in the study of Tropical Medicine.] (Note made during correction of the proof-sheets.)

REFERENCES.

1. Trans. R. Ac. Med., Irel., vol. xx, 1902.
2. Arb. a. d. kaiserl. Gesundheitsamte, Bd. xix, 1903.
3. Centralbl. f. Bakt, 1893.
4. *Loc. cit.*, p. 571.
5. Zur Kenntniss der Darmamöben und der Amöbenenteritis. Veröffentl. aus dem Gebiete des Militär-Sanitätswesens, Heft 20, 1902.
6. Journal of Hygiene, vol. iv. p. 508.
7. *Loc. cit.*, Bd. xx, p. 169.
8. Schaudinn, *loc. cit.*, Bd. xviii.
9. Thompson Yates' Reports, vol. v, 1903.
10. Conférence faite au Conseil National d'Hygiène le 19 Mai, 1901 (Assuncion 1901), and Ann. Pasteur, 1903, p. 241.
11. Ann. Pasteur, t. x, 1896.
12. Journ. of Comp. Path. and Therapeutics, vol. xvi, 1903.
13. Journ. of Tropical Medicine Dec., 1902, and Thompson Yates' Reports 1902, vol. iv, part 2.

14. Letter dated Uganda, 5th April, 1903, and addressed to the Royal Society, "B. M. J., 1903, vol. i, pp. 1218 and 1431.
15. Reports published by the Royal Society in 1903, B. M. J., 21st November, 1903.
16. B. M. J., 1904, vol. ii, p. 650.
17. Journal of Infectious Diseases, vol. i and ii, *passim.*
18. Generations. und Wirthswechsel bei Trypanosoma und Spirochæte, Arb. a. d. kaiserl. Ges., Bd. xx, 1903.
19. Trypanosomiases, Paris, Masson, 1904.
20. Abstr. in Centralbl. f. Bakt., Bd. ix, 1891, p. 120.
21. Ueber Malaria, und andere Blutparasiten, etc., Jena: Fischer, 1898.
22. Ann. Pasteur, 1903.
23. Comptes Rendus de la Soc. de Biol., 20th April, 1903, No. 10.
24. Centralb. f. Bakt, 1901, Orig., p. 569.
25. Arb. a. d. k. Ges., Bd. xx, 1903, p. 438.
26. Journ. of Hygiene, 1905, No. 1.

Discussion on Dr. E. J. McWeeney's Paper.

The President: I think the most fitting way of proceeding, after listening to Professor McWeeney's most interesting Paper, which I am sure we have all heard with the greatest pleasure, is to ask Sir Patrick Manson to open a discussion upon it. The Paper deals with points which are highly technical and extremely interesting, but not all of us have had an opportunity of pursuing such studies as would place us in a position to open a discussion of such questions. I will therefore call upon Sir Patrick Manson.

Sir Patrick Manson: Sir, I have no doubt you will agree with me in thinking that Prof. McWeeney has conferred a great boon not only upon this Society, but upon all who are interested in questions bearing upon this particular subject of Protozoa in relation to disease. Schaudinn's Paper we have looked at, but, I fear, very few of us have read it. The subject is abstruse and difficult, and the German in which it is discussed is more abstruse and difficult still. I have read one or two translations, but the translations themselves require to be translated before they are intelligible; but now, with Professor McWeeney's able disquisition in our hands, no one has any longer an excuse to remain ignorant on this subject.

These observations of Schaudinn are exceedingly important, and I presume they are correct; but they are not without opponents. Only lately McNeal and Novy, two American protozöologists who have given a great deal of time and attention to the study of Trypanosomes, have ventured to criticise Schaudinn's conclusions, particularly as regards the connection of Trypanosomes with Halteridium and similar parasites. They say distinctly that

Schaudinn has made a mistake. I do not say so; it is they who say so. They say that he has been working with mixed infections, and not with pure Trypanosoma infections. They say that in certain birds in America—or at all events in a large proportion of them—they can by a process of cultivation, demonstrate the presence of Trypanosoma in the blood, whereas in only a small proportion they can detect anything in the nature of Endocorpuscular Protozoa. They contend that Schaudinn has been working with birds in which a Trypanosoma infection was superadded to another and independent infection by Endocorpuscular Protozoa. That is their criticism, and not mine.

We have had a great deal of information from Professor McWeeny this evening: I should like a little more. Schaudinn tells us he divides the öokinets into three kinds; an "indifferent," a "male," and a "female." I should like to know why he calls them indifferent, male, and female. These öokinets are themselves the result of a sexual process: the microgamete and the macrogamete are the male and female, and the result of the union is the öokinet, which shows no evidence of possessing sexual qualities. Why, then, class them male and female? I have no doubt there is an explanation of the use of these terms, and I should be grateful if Professor McWeeney would supply it.

As regards the important communication on the subject of the Amoeba of Dysentery —*Amoeba histolytica*— I would utter a word of caution as to concluding that this matter is completely settled. The modern doctrine which divides the dysenteries into two categories, those produced by the Bacillus of Shiga and those produced by the *Entamoeba histolytica*, is very complete and fascinating, but it does not square at every point, at all events in my experience, with clinical observation. Nor does it seem to logically fit in with the facts of the case. For example, Schaudinn states that the cause of tropical Dysentery is this *Entamoeba histolytica*, which, in its vegetative form, exists and is active in the bowel of dysenterics during the acute and active phases of disease, but which, when the disease begins to subside, forms spores which become encysted; and, he contends, it is by these spores that the disease is propagated and spread from one person to another. Now, why should this be if the disease is a tropical disease only? Why should these spores not be capable of existence outside the human body, and produce disease in non-tropical countries as well as tropical countries? In view of the resistive

properties of an encysted spore, one would be inclined to think that the disease it gave rise to would, as regards its geographical distribution, behave like bacterial diseases and not as those of a Protozoal origin. Any disease that is limited geographically, like tropical Dysentery, depends for its spread upon a medium which conveys the infective agent from the sufferer to the hitherto non-infected person; and this medium or transmitting agent is the factor that determines the geographical limitations of the disease. But in this instance of Amoebic Dysentery, there is no limitation as regards the transmitting agent: the spore falls on the ground and is directly communicated, unaffected by temperature, to another person. Why, therefore, should not that disease be a pandemic disease as well as a tropical disease? Schaudinn's views, to my mind, do not get over this difficulty.

There is another point I should like to say a word or two about, that is: Schaudinn (or Professor McWeeney) assumes in the case of Leishman's body that the transmitting agent should be, or must be, an anthropophagous insect—a blood-sucker of some sort. I do not quite see why there should be an insect or other kind of blood-sucker. For we now know as a fact that the Leishman body is extruded from the human body by ulcerated surfaces. Patients who suffer from Leishman's disease die, as a rule, from some form of ulceration of the intestine, and from these ulcerated surfaces the Leishman body escapes into the outer world; so that it is more likely that a non-blood-sucking insect conveys the parasite from the fæces or ulcerated surfaces to an uninfected individual. I think this idea is borne out by another circumstance in connection with the bodies referred to. An exactly similar, or possibly the same, body is found to be at the root of the disease known as Oriental Sore, in which case the parasite must escape from the ulcer on the surface of the body, and not by the blood.

Some time ago, possessed with the idea that Leishman's body was possibly a blood parasite, I tried in one case to see if the usual absence of the parasite from the blood might depend on some circumstance connected with light or darkness, day or night; and I instituted a series of observations upon a patient suffering from the disease, to see whether the blood might not, at some period of the 24 hours, contain Leishman's body. My observations were negative: I never saw any bodies during the day or night in the blood. These observations were confirmed by Dr. Low, of the Tropical School. So far, we have no

evidence to show that Leishman's body is in any circumstances a blood parasite.

The idea that Syphilis may be one of these Protozoal diseases is an exceedingly fascinating one; and I think that there is some ground to hope that in these Leishman's bodies we have a clue to that mysterious infection.* Some years ago, a medical man who had practised in Persia and Arabia was invalided home in consequence of a severe attack of Oriental Sore. He came to England, and whilst still suffering from the Sore, he developed what was believed to be tubercular disease of one testicle. The testicle was excised, with the idea of preventing general tuberculosis. The disease appeared, however, in the other testicle, which was also excised. I saw the patient about that time, and the idea that presented itself to my mind was that the Oriental Sore was a sort of Chancre, the point of entrance of a poison which became diffused, and afterwards produced secondary or tertiary effects, one of which was a specific infection of the testicle. Whether the idea is a correct one or not, I cannot pretend to say. It is, at all events, one that suggests a fresh field for observation.

DR. COPEMAN: I am obliged to you for calling upon me, for the reason that it gives me the opportunity of adding my thanks to those of Sir Patrick Manson for this admirable and interesting address; but I think it will be generally admitted that it is impossible for anyone who has not done original work on the subject of Protozoa to adequately discuss the Paper. It is, however, as far as I am able to judge of it, a most interesting and most judicial Paper. It sets out, in a delightful way, the work of other observers, and more particularly of Schaudinn. It affords those who have been unable either to work at the subject or to see the original Papers an opportunity of learning what has been done on the subject, and perhaps, in course of time, after carefully reading through the Paper again, of criticising to some extent the points brought forward.

Professor McWeeney states that the Paper contains no original research of his own; but, at any rate, it contains several most interesting suggestions which I take to be his own. There was one that particularly appealed to me because for some years in my leisure time I have worked

* [See footnote on p. 103. Schaudinn's discovery of Spirochætæ in Syphilis was not made at the time when Sir P. Manson used these remarkable words.]

on the subject of sex; and that was the suggestion as to the part played, on the one hand, by the additional Spermatozoa, and by the "polar" bodies on the other hand. I have often wondered what was the reason for the enormous number of Spermatozoa which are always produced in mammalia, when we know that, as a general rule, only one can be utilised in fertilisation; and I have often wondered also what becomes of the "polar" bodies, which are always extruded previous to fertilisation. No suggestion, so far as I am aware, has previously been put forward as to what part may be played by these bodies, or by the excessive amount of material which is cast off from the cells of the seminiferous tubules and other parts of the genital track. One knows that Nature is always prolific; but I agree that it is highly probable that this excess of male and female elements, as well as the fluid portion of the secretion, do play some particular part in the process. I think Professor McWeeney's explanation is a very ingenious one, and one which I think may not improbably turn out to be correct.

I am glad also to see that Professor McWeeney, in his historical statement as to the investigation of the Trypanosomes connected with diseases in man, gives a special place to Castellani: because, I think, it has of late been the custom hardly to give him a fair position in the matter of discovery. One knows that perhaps he did not at first attach a large amount of importance to the presence of Trypanosoma in the cerebro-spinal fluid; still, he did demonstrate its presence, and it is quite possible that unless he had made his examination of the cerebro-spinal fluid, the actual discovery of the Trypanosoma of sleeping-sickness might have been considerably delayed. Whilst others, like Bruce and his colleagues, really brought the discovery to a definite conclusion, I think—as I have said—that had it not been for Castellani's previous work, that of other investigators might not have been so fruitful as it has been.

As regards Professor McWeeney's suggestion of the ultra-microscopic and so possible *filtrable* nature of the specific organism of small-pox and vaccinia, I think that this is definitely disproved by my experimental work (an extension of that of Chauveau and Burdon Sanderson), published in the *Transactions* of the London Congress of Hygiene and Demography in 1891.

I desire once more to thank Professor McWeeney for his Paper, which I shall hope to read again at my leisure, as it is hardly possible to follow such a multitude of new facts in the space of time occupied in reading the Paper.

SIR PATRICK MANSON: I should like to add a few more words. In one paragraph, towards the end of the Paper, Professor McWeeney says that a new form of Spirillosis in the human subject has been discovered by Dutton and Todd on the Congo. That disease has already been discovered by the bacteriologist of the Uganda Government, Dr. Ross, and had been linked up with the Ornithodorus a considerable time before.

There is another question I should like to ask. Did Koch succeed in infecting his birds with Halteridium by injection of Halteridium blood, or was it in a more indirect way? I always understood that other Trypanosomes could be inoculated directly, but that Halteridium could could not be directly inoculated.

THE PRESIDENT: Before calling upon Professor McWeeney to reply, I want to offer him, on behalf of the Society, our most cordial thanks for his admirable Paper and demonstration. We are, indeed, very grateful to him for coming so far to teach us so much.

PROFESSOR MCWEENEY: The observations which have been made, Sir, by yourself and other speakers have been a source of great pleasure to me. It is true that I have come a considerable distance to read this Paper; but the occasions on which an Irish worker has been asked to read a Paper before a London Scientific Society have been few and far between; and accordingly I regard it as a great compliment and a very great honour to be asked to come here and deliver this Paper, and the way in which it has been received has been a source of still greater pleasure to me.

The remarks made by Sir Patrick Manson, whose researches and discoveries in this field of observation have gained a wide-world reputation, and give him a special authority to speak on the subject—what he has said in confirmation of the general views which I venture in accordance with Schaudinn to express, has been eminently satisfactory to me. Eminently satisfactory is it to learn from him that in the main the criticisms, apart from those of Novy and McNeal (which, by the way, had escaped me when I was reading up for this Paper—had I seen them I would have dealt with them in the Paper) are favourable to Schaudinn. It is highly satisfactory to find this, because I am bound to say that, after very careful perusal of Schaudinn's papers (I have read all that he has written upon the subject), they give me the idea of having the true

ring of genuine accuracy and research, and of being very full and accurate. He has written another Paper on the Tertian parasite, which is full of the most beautiful drawings; and the whole work gives one the idea of completeness and painstaking accuracy. At the same time, it should have been remarked that I did not give in an absolute adhesion to all his conclusions. I have but pointed out what appear to me to be some of the more important inferences from his discoveries.

The question which Sir Patrick Manson has asked me undoubtedly draws attention to what appears to me an unjustifiable part of Schaudinn's nomenclature. He speaks of the three kinds of Trypanosomes as "indifferent," "male," and "female." It is quite true they cannot be so regarded, since they are themselves the immediate result of a sexual process. Schaudinn has not exactly stated, so far as my recollection goes, why he selected these terms. His general view is that the "male" Trypanosome is in its mode of formation and morphology precisely similar to the microgamete, and that what he calls the "female" Trypanosome resembles the macrogamete in having dull protoplasm and a relatively small nucleus. That, I think, is the current of thought which has suggested this nomenclature to Schaudinn; but whether it is justifiable or not is a point on which I should be inclined to follow Sir Patrick Manson, who is in a better position than I am to form an opinion on such a point.

With regard to the question of the *Amoeba histolytica*, so far as I have been able to gather, Sir Patrick Manson's difficulty is this: if the parasite is not limited to tropical countries, neither ought the disease to be so confined.* As to this I can only offer a suggestion of my own: might not the ripening of the cysts be a question of temperature? Would not that account for the non-appearance of the disease in some climates? They might require a high temperature to mature in, and such a temperature might only occur where this type of disease is endemic.

In connection with what Sir Patrick says as to the Leishman body not being conveyed by a parasite—that is just the value of discussions like these—the value for me as a man removed from the actual field of observation. I figured to myself a parasite in the liver or the spleen, etc., and I

* Neither the parasite nor the disease is, as a matter of fact, so limited. In addition to the cases of Amœbic Dysentery reported by Jäger (*Berl. klin. Wochenschr.*, 1901), Albu, quite recently, records a similar case that ended fatally. The disease was contracted in Breslau (*Ztschr. f. Klin. Med.*, Bd. lxv, p. 433).

did not know it was excreted from the sores, and therefore capable of being conveyed by other than blood-sucking parasites.

With regard to the disease described three or four months ago under the name of Human Tick Fever, the point of interest appeared to me to be the announcement made by Major Ross, in the *British Medical Journal* last Saturday, that it was due to a Spirillum, and was propagated by the bird-tick, Ornithodorus. That, of course, brings it at once into close connection with the Piroplasmic diseases, which are always produced by ticks.

The last question of Sir Patrick Manson was whether Koch succeeded in producing Halteridium by direct inoculation. I am under the impression that he did. His Paper on the subject, which is contained in the *Zeitschrift für Hygiene* of 1899, describes the results of his study of Halteridium and Proteosoma in the blood of birds. It is a very curious thing that he should never have come across the Trypanosome stage. [My recollection was at fault here. On referring to Koch's Paper, I find that, although he succeeded with Proteosoma, he failed to communicate Halteridium by direct inoculation.]

I am extremely grateful to Dr. Copeman for all he was good enough to say of the Paper. It is quite true that it would require to be read over several times in order that it might be criticised, because I was obliged to compress a great deal of matter into as few words as possible. Coming from one who has done such good original work as Dr. Copeman has upon the virus of Small-pox, such commendation is very agreeable; and it would be extremely interesting if Dr. Copeman could carry out some Small-pox work on the lines indicated. The virus might possibly prove to be a filtrable one, and of Protozoal nature.

I was very glad to be able to lay stress on the work of Castellani, whose application of the centrifuge to the examination of the cerebro-spinal fluid seems to be the critical point in the whole pathology of Sleeping Sickness: demonstrating as it did the constant presence of the Trypanosome at a period when the researches of the Portuguese Commission tended to ascribe to a Coccus the leading *rôle* in the ætiology of the disease.

I think nothing now remains for me but once more to thank the Society for the patience with which they have listened to me.

ANKYLOSTOMIASIS.

By A. E. BOYCOTT, M.A., M.D., B.Sc.

(*Read: Friday, March 17th,* 1905.)

In trying to combat the ravages which are produced by any of the various infective parasitic disorders to which man is exposed, it is of the first importance that the sanitarian should be in possession of full information as to the life-history of the parasite which is the causative agent, and especially that part which is spent outside the human body. Without such knowledge, it is difficult to plan, still more difficult to put into effect, any devices for the undoing of these parasites other than those of an altogether exploratory or empirical nature. Some parasites, such as the *gonococcus*, have, we believe, no saprophytic existence, and no other host than man; remedial measures are perhaps neglected, but they are none the less simple and obvious. In other instances, such as yellow fever, our knowledge of the physiology of the life of the causative agent has been gained in advance of our acquaintance with its morphological nature; it is, however, sufficient to give us good expectations that material progress in the suppression of the disease may continue to be made. In yet other cases—and this class comprises the majority of infective disorders—we know practically nothing of the life of the parasite outside the body of man, and perhaps of certain animals, and any efforts which may be directed against the *B. typhosus* or the *pneumococcus* are necessarily, to a large extent, of the nature of vague experimentation.

The greater part of mankind live in the tropical and sub-tropical parts of the globe. Taking the world as a whole, the two fundamental diseases to which they are here subject are malaria and ankylostomiasis. Until the history of the malarial parasite in its life outside the human body was known, the only weapon in our possession was at once empirical and purely defensive; those great researches of Ross have enabled us to plan a philosophical attack in a practical and, we hope, an efficient manner.

Our knowledge of the morphology and physiology of the

life-history of *Ankylostoma* is still far from complete in many details. In the main outlines, however, it is sufficient to enable us to trace a close connection between the habits and mode of life of the parasite on the one hand, and the whole range of its pathology on the other, and it is from this point of view that I should like to consider the subject of my paper. In the first place, I propose to briefly consider the life-history of *Ankylostoma*; then to review the conditions which may influence this history; and, finally, to consider the applications of these facts to sanitation.

The full-grown sexual *Ankylostoma* inhabits the upper part of the small intestine in man. There are two known species—one, known as *A. duodenale*, is found in Europe, Egypt, etc., while the other, *A. americanum*, has recently been differentiated from the old-world species by Stiles in the United States. The details of the life-history of this latter species have not been fully worked out, but, as far as our information goes, it does not differ from that of the older species, and the effects produced seem to be identical. In case, however, further research reveals any important differences, it is as well to say that the following account refers to *A. duodenale*. The worms are found in the intestine in numbers which vary from two or three to many hundreds. The females lay eggs in very large numbers, and in consequence the stools of an infected individual are often crowded with eggs.

As seen in fresh fæces, the eggs are characterised by a thin, clear, colourless shell, about 60 μ by 40 μ, of a regularly oval outline. The greyish contents are easily seen, and the egg when first passed consists as a rule of four segments. Under suitable conditions, segmentation proceeds very rapidly, and a morula stage is soon reached. The egg may contain an active vermiform embryo in 18 hours, which shortly escapes from the shell. The newly-hatched larva is about 0.27 millimetres long. In favourable surroundings growth proceeds rapidly, and in another 24 hours the larva has moulted, and attained a length of 0.55 millimetres. After a second moult, the beginnings of a definite "chitinous" sheath can be readily made out; and a third moult brings the larva to a final measurement of about 0.6 millimetre. The sheath is now very well developed, and to a certain extent, especially at the head and tail, the larva is free to move within it. This stage is known as the "encapsuled" or "full-grown" larva, and is one of the fundamental landmarks in the cycle of development, representing as it does the adult saprophytic phase of

the parasite. So long as it does not gain entrance to the human body, no further growth takes place; the larva remains asexual, and no multiplication of individuals occurs. If, on the other hand, the larva can obtain a lodgment in the human intestine, development soon proceeds; rudimentary sexual organs appear, and after a series of moultings the adult parasitic stage is reached, with sexual differentiation into male and female individuals, and a rapid multiplication of individuals.

The important points in this life-history are, firstly, that there is no intermediate host, and, as is usual with these higher parasites, the infection of each host is specific for each parasite: thus a dog *Ankylostoma* cannot infect man, nor a human *Ankylostoma* a sheep. In the second place, there is no multiplication of individuals, either within or outside the host. Such increase in numbers can only be brought about by a due and alternating succession of parasitic and saprophytic phases.

The statement, originating, I believe, with Giles and since repeated by several observers, that *Ankylostoma* has a sexual stage outside the body, are to be received with great caution. Their results are readily explained by the assumption—perhaps unjustifiable—that they were not dealing with pure cultures. There are a number of small, entirely saprophytic, nematode worms which are of very general occurrence in earth. The eggs and larvæ of some of these closely resemble those of *Ankylostoma*; the adult sexual forms are, however, quite different, and appear to be exactly similar to the worms figured by Giles and others. These earth nematodes flourish and multiply to an extraordinary extent in fæces to which they may gain access; and I have found them in such situations, as well as in uncontaminated earth, in many English mines, both coal and metalliferous, infected and non-infected. Their frequent presence, and the close resemblance which the larvæ of some of them bear to undoubted *Ankylostoma* larvæ, vitiate also the value of observations on the occurrence and habits of (so-called) *Ankylostoma* in its natural saprophytic habitat. On the experimental side, numerous experiments have, in my hands, given uniformly negative results, and in no case has any sexual form been observed in cultures made from fæces which had not been exposed to contamination from the earth. Similar cultures made from material containing *Ankylostoma* eggs, which had been picked off the ground, showed on many occasions sexual forms of small nematodes belonging to the family *Anguillulidæ*, but only

the larvæ of *Ankylostoma.* Many different methods of culture were tried—aerobic and anaerobic—with duodenal mucosa and various extracts made from it, with blood, at different temperatures, in acid and alkaline media, etc; but genuine *Ankylostoma* larvæ could never be persuaded to make any further advance in development from the encapsuled stage.

The chances of multiplication are further diminished by the fact that it is only at a certain point of the saprophytic development that *Ankylostoma* becomes capable of infecting its host. It was clearly shown by Leichtenstern that the eggs and young larvæ are not infective; he found that eggs appeared in the stools only after the full-grown encapsuled larvæ had been administered to the subjects of experiment. I would direct especial attention to this point, which is of unique importance in the consideration of the distribution and epidemiology of the disease.

The time-relations of these changes are also of practical importance. From the fresh egg to the encapsuled larva takes at the least four or five days, and usually longer. The second period of growth, within the host, from the encapsuled larva to the sexual adult, occupies about five weeks. The possible length of life at each stage is less well defined: if the eggs are exposed to conditions which are unsuitable for their immediate development they do not live very long—four or five weeks, perhaps, often only a few days. The encapsuled larva is capable of a very prolonged saprophytic existence. I have been able to keep such larvæ alive in the laboratory at room temperature for more than twelve months. During the whole of this period they lived in an ordinary Petri dish, with no more nourishment than that contained in the fragment of fæces from which they were originally hatched, and in the London tap-water which was added from time to time to make good the loss by evaporation. In their natural habitat they are probably capable of living for a still longer period. The adult parasitic form is also long-lived. Stiles found several cases of infected children in a home in the United States, which was situated outside the endemic area. They had been brought from an infected district more than six years previously, and it is not possible to believe that they had been re-infected during their residence in the well-conducted institution in which they lived. Another interesting example of the duration of the infection came to the notice of Haldane and myself in Cornwall. A youth worked in the infected area—which is here strictly confined to the

underground workings of the mines—in 1898; he soon became anæmic, and gave up the employment. When we saw him at the end of 1902, he had spent the intervening four years in driving a butcher's cart, and had not been even to the surface workings of a mine; yet we were able to find a few eggs in his stools.

A knowledge of the paths of infection gives us little assistance in our endeavours to suppress the parasite. It has been proved long since by direct experiment that the mouth affords for *Ankylostoma*, as for other parasites, an easy mode of access to the intestines; and, until recent years, this was believed to be the only path of infection. Looss, and after him several other observers, have shown, however, that the encapsuled larvæ are capable of penetrating the unbroken skin *viâ* the hair-follicles. It is stated that they are then carried by the blood stream to the lungs, where they form emboli. Gaining access in this way to the air passages, they pass up the trachea and down the œsophagus, and so find their home in the small intestine. If any part of the surface of the body may thus afford a possible point of infection, it is clear that no direct indication is afforded in the matter of prevention; and the brilliant researches which have led to this notable addition to our knowledge are scarcely other than depressing to the sanitarian.

There is, I think, no reason to doubt that infection may, and does, occur by direct reception of the larvæ into the stomach, and thence into the small intestine. Experimentally, encapsuled larvæ may survive for two or three days in strong mixtures of pepsin with hydrochloric acid up to 0.5 per cent., and in alkaline trypsin solutions. If swallowed larvæ pass through the walls of the pharynx or œsophagus, in order to undergo development before facing the ordeal of gastric juice (which is by no means always present in the stomach), it is difficult to see why the interval between infection and the appearance of eggs in the stool should be so much longer in a skin (70 days) than in an alimentary infection (30 days). My own experience is that, 18 months after having made entirely ineffectual attempts to produce an infection through the skin, I became infected at once through the mouth after an accident with a pipette.

We have next to consider the influence of various external conditions on this cycle of development.

1. *Temperature.*—The eggs are killed in about twenty-four hours at a temperature of 40 deg. Cent. (104 deg.

Fahr.), or at a nearly freezing temperature. They may hatch at any temperature from 16 deg. Cent (61 deg. Fahr.) to 38 deg. Cent. (100 deg. Fahr.); in an ordinary hot incubator (36 deg. or 37 deg. Cent.), the eggs often die without hatching, but a few larvæ generally hatch out in about twenty-four hours, and then rapidly die away. The optimum temperature would appear to be about 24 deg. Cent. (75 deg. Fahr.); at rather higher temperatures the eggs hatch more quickly, but the mortality among the young larvæ is high, and many fail to reach the encapsuled stage. The determination of the lowest temperature at which complete development may take place is a point of the greatest practical importance. Below 13 deg. Cent. (15 deg. Fahr.) the eggs will certainly not hatch; but I have twice succeeded in hatching the eggs and growing the larvæ to the encapsuled stage, at a temperature which was generally 15.9 deg. Cent. (60.6 deg. Fahr.), and never exceeded 17 deg. Cent. (62.5 deg.) I may mention that these experiments were made in a Hearson incubator, and that the temperature was noted carefully at very frequent intervals during the whole observation. Several lots of eggs failed to hatch at this temperature, or if hatched failed to develop to the encapsuled larvæ, and there is no doubt that such a low temperature is by no means favourable to *Ankylostoma*. A free and full development is, I think, seldom shown at temperatures below 20 deg. Cent. (68 deg. Fahr.).

Temperature has also a very marked influence on the rate of development. At 35 deg. Cent. free larvæ may appear within twenty-four hours, and encapsuled larvæ in four days; while at 16 deg. Cent. the eggs may take three to seven days to hatch, and encapsuled larvæ may not be present for a fortnight.

Once the larvæ have reached the encapsuled stage, they are relatively indifferent to temperature. I have never succeeded in keeping them alive for more than a few days at 37 deg. Cent., while at room temperature (10 deg. to 20 deg. Cent.) they live for many months. They appear to live equally long at 25 deg. Cent.

2. *Moisture.*—A certain amount of moisture is necessary at every stage, and "ordinary" drying at room temperature kills eggs and larvæ in an hour or two. On the other hand, excessive wetness is fatal to the eggs. Infected fæces covered with a layer of water ½ in. in depth will never hatch at any temperature, and die in about a

week.* The larvæ, on the contrary, prefer to live in water, and the most succesful method of hatching which I have tried is one in which the fæces are kept damp, but not submerged, while they are surrounded with a moat of water into which the newly-hatched larvæ may escape.

3. *Oxygen*—These differences in the relation of *Ankylostoma* to water at different stages of development are explicable, I think entirely, by variations in oxygen requirement. The eggs will only hatch in the presence of a very free supply of oxygen. A gram of infected fæces corked up in a 20 cc. test tube fails to show any larvæ after exposure to a suitable temperature. The inhibitory action of water appears to depend on the relative exclusion of oxygen, for if air be bubbled through a mixture of infected fæces and water at 25 deg. Cent. larvæ hatch out (though only in small numbers) in three or four days. If hydrogen is bubbled through a similar mixture, no development takes place, so that the result obtained with air is not due to the mechanical disturbance. The encapsuled larvæ, on the other hand, show anaerobic capacities of an order which is somewhat remarkable in an animal so high in the zoological series. They will live and exhibit the most lively movement for three weeks in an atmosphere of hydrogen at 17 deg. Cent., and for at least eight days in carbon dioxide, or in a vaccum.

Oxygen is, then, necessary during active growth, but not during the resting stages. Experiments in the anaerobic development of the larvæ showed that development did not proceed unless oxygen was present, though the young larvæ were not immediately killed. These facts are in complete accord with the observed inability of the eggs or young larvæ to produce an infection if taken into the alimentary canal.

4. *Chemical Agents.*—The most exaggerated views have been held on the supposed extraordinary powers of resistance to antiseptics exhibited by *Ankylostoma* larvæ. It is entirely a question of the time factor. It is quite true that the encapsuled larvæ may exhibit active movements for a quarter of an hour after immersion in a 4 per cent. solution of corrosive sublimate, but they were surely killed in four hours in a 0.1 per cent. solution of sublimate, 0.25 per cent.

* After repeating this experiment a number of times with the uniform result mentioned above, I have lately observed an exception in which a few dead one-day larvæ were found in a preparation which had been continuously covered with two inches of water at room temperature.

izal, or 1 per cent. carbolic acid. In twenty-four hours, 0.1. per cent. izal or 4 per cent. sodium chloride will kill the adult larvæ. The explanation of the marked resistance shown by these larvæ when first exposed to the action of the poison is due to the presence of the thick sheath, and it is only after a certain time that this protection is penetrated. Young larvæ, with a much thinner coat, are more easily killed, and in this way the application of a 2 per cent. solution of sodium chloride to infected fæces will prevent the development of encapsuled larvæ, though 6 per cent. is required to prevent the eggs hatching, and 4 per cent. to kill the encapsuled stage.

Bruns found that the more he diluted fæces with water, the quicker and more complete was the development of larvæ. All observers have noticed that occasionally no larvæ can be obtained from fæces full of eggs. This seems to be due to the presence of some substances in certain stools which kill either the eggs or young larvæ. I have found that aqueous extracts of some fæces will kill the newly-hatched larvæ. Why this is I do not know. It is not a question of the reaction. Bruns' view that it is due to the plasmolysing effect of salts can hardly be correct, since fæces contain hardly any soluble salts.

5. *Light.*—Sunlight is said to be quickly fatal to the larvæ. My own experiments on this point gave entirely negative results, but the only sunlight available was that which may be had in London.

It will now be necessary to consider shortly the relation of the infected individual to the infecting agent.

The typical result of *Ankylostoma* infection is an anæmia of greater or less severity. Definite data on the length of time after infection which must elapse before well-marked symptoms are produced are not available, but there are fairly clear cases in which a very profound degree of anæmia has been reached within three months of infection. In the intervening period symptoms are indefinite, but certain "dyspeptic" pains without any association with the taking of food are often present, and have, I think, a definite connection with the presence of the parasite in the small bowel. In many cases a skin affection (botches, bunches, gourmes, mazamorra, coolie-itch, etc.) of varying severity will be present. The degree of anæmia varies from a diminution of 10 or 20 per cent. in the percentage oxygen capacity of the blood, to a condition of the blood which is not exceeded in severity even in the worst cases of perni-

cious anæmia. Patients in the latter condition are, of course, helpless, and frequently die unless treated. Even the former class may exhibit some symptoms of dyspnœa on severe exertion, and their working capacity is thereby distinctly—though perhaps only slightly—diminished. Many cases seem to reach a condition of moderately severe anæmia, beyond which they do not sink. With a percentage oxygen capacity diminished by one-half, they may drag on for years in a state of constant ill-health and inability to do their share of work. The actual destruction of life caused by *Ankylostoma* is considerable enough, but the chronic illness which ensues in neglected and unrecognised cases is responsible for far more unhappiness.*

One important point in the pathology of infective diseases is illustrated particularly well by ankylostomiasis. This is, that many of those who harbour the worm suffer from no symptoms of disease at all. The percentage of sick people in a series who are about equally exposed to an infection varies a great deal with different infections, as well as with different series of possible subjects. From the demonstrated infection of 2, or 5, or 10 per cent. of a population exposed to water- or milk-borne enteric fever, there is every gradation under varying circumstances to the destruction of half a population by plague, sleeping-sickness, or kala-azar. Now, in these instances, we are to a very large extent compelled to judge of the infection of the individual by the obvious presence or absence of disease. In *Ankylostoma*, however, we have a parasite of such dimensions that the criterion of its presence may, if necessary, be made entirely macroscopic, and we can ascertain very clearly what proportions of a population are on the one hand infected, and on the other hand suffering from definite disease. It has been found that in localities which are especially favourable for the worm, practically every individual who is exposed to infection actually harbours the parasite. Dobson found eggs in the fæces of 75 per cent. of the natives in Assam; and in one of the Cornish mines, after examination of about a quarter of the underground hands, taken at random, I failed to obtain satisfactory evidence that a single man was without the worm. Yet, at the worst, not more than 10 per cent. of the men were actually ill, and the great majority expressed themselves

* The truly appalling state of a population saturated with neglected *Ankylostomiasis* is shown very well in the *Report of the Commission for the Study and Treatment of "Anæmia" in Porto Rico*, by Ashford, King, and Ingaravidez. San Juan, P. R. 1904.

to be in perfect health. Of 21,612 men who were found to have eggs in their stools and received specific treatment in Westphalia, only 6.8 per cent. showed definite anæmia. These considerations, I think, support the suggestion that the apparent partial incidence of many infections depends more upon the resistance of the infected individual than upon the mere inclusion of the parasite within his body.

One has, then, to distinguish two classes of infected persons—those who have *Ankylostoma* infection, and those who have the disease ankylostomiasis. Without entering upon a purely pathological discussion, it is perhaps worth mentioning that the tissues of the former class—who may be called immune—are not absolutely indifferent to the presence of the worm, since the blood in these cases shows, as I know from personal experience, the somewhat characteristic changes in the leucocytes to a degree which on the whole is greater than that found in those who are actually anæmic.

The variation seen in the degree of illness produced is, then, due in part to variation in the natural resistance of the individual to the toxic products of the worm. It is also due to variations in the magnitude of the infection. Cases are well established in which a fatal issue has ensued upon the presence of comparatively few worms in the bowel. Perfect health, on the other hand, is not inconsistent with the presence of hundreds of parasites. There is, however, some relation between the degree of anæmia and the amount of infection, and especially the frequency of infection. Individuals exposed to infection under conditions which are eminently favourable to the worm on the whole suffer more severely than those who only meet with the extra-corporeal stage where it has to struggle for existence. These considerations are in harmony with the fact that the anæmia is not due to the abstraction of blood by the worms. This has been clearly established by the investigation of the blood-volume of Cornish patients by Haldane, and is supported by many other details of the infection.

Such is a brief account of the life, habits, and effects of *Ankylostoma*. From these data it is possible to deduce a satisfactory, if not an entirely complete, explanation of most of the facts which are the result of epidemiological observation rather than that of laboratory experiment. Since both lines of investigation lead us to the same ends, it is unnecessary—as it would perhaps be

undesirable—to consider which of the two gives us the better indication for practical hygiene.

Ankylostoma may infect either men or places. The infection of men is immediately the concern rather of the clinician and the pathologist than of the sanitarian. There are, however, two points which require emphasis. I have already laid stress on the fact that many individuals harbour the worm without showing any signs of obvious illness. Yet such persons are potential carriers of infection to other places, no less than those who are actually ill. The healthy "worm-carrier," indeed, is the greater factor in the dissemination of the worm by his more facile locomotion. Such people, too, may remain infective for years after they have removed from an infected locality. The parasite may thus travel all over the world in the persons of those upon whom no suspicion would be likely to fall, and who would, in most instances, be themselves entirely in ignorance of their dangerous character. On the other hand, an infected individual cannot spread the infection before his own parasites have commenced to lay eggs. This favourable point is more than counterbalanced by the fact that, since the diagnosis in the ordinary way rests on the discovery of these eggs in the stools, he would pass even a specific medical examination during the same period.

For the infection of places, three conditions are obviously necessary. Firstly, that infected fæces should be deposited; secondly, that the place so contaminated should present those combinations of temperature, moisture, and æration, which have been already defined as necessary for the development of the infective stage of the worm from the egg. And thirdly, that an interval of time should elapse between the deposition of the eggs and the development of the infectivity of the place: as, however, this interval may be so short as four or five days, this condition does not come to be of any greatp ractical importance, though it precludes a constantly-recurring cycle of auto-infection.

It is, of course, possible that the encapsuled larvæ might be carried from place to place as such. Under these circumstances, conditions of temperature, etc., play a much less important part. The inability of the larvæ, however, to withstand drying renders this mode of transmission of no practical importance, except over very short distances.

The first condition, that fæces should be deposited in such positions that human beings shall thereafter come into contact with them, is fulfilled all the world over with varying degrees of completeness. The majority of man-

kind makes no special provision whatever for the disposal of excreta. Among relatively savage races this is perhaps not unreasonable; but it is surprising to learn that among, *e.g.*, the farming class in the southern parts of the United States, even the most elementary privy accommodation is unknown. With permanent houses the result is obvious, and the whole of the soil in the immediate neighbourhood of their dwellings is saturated with fæcal contamination, growing worse and worse as time goes on. And it is just these very parts of the earth with which the inhabitants come into intimate daily contact in the pursuit of agriculture. A nomadic people are in a far better position, as their filth is periodically left behind them, and the custom among the Burmese of having a fresh capital for each king finds ample justification in considerations of public health alone. Greater care is taken in the disposal of excreta in this country, I suppose, than in any other; yet a brief inspection near any large centre of population will soon show anyone whose observation is sharpened by interest that there is abundant opportunity for the careless or unwary to come into contact with human excrement. That the population of London have not infrequent contact with stale human fæces is shown by the fact that the eggs of the worm *Trichocephalus dispar* were found in the stools of rather more than 10 per cent. of 250 patients taken at random in the general wards of Guy's Hospital. Dr. French is still continuing this inquiry, and I am indebted to him for permission to mention the main result now. It is one of great interest, for in *Trichocephalus* we have a parasite which is not capable of any multiplication outside the body, and one, too, which (as far as is known) can only become established in the bowel by ingestion with the food of eggs which have lain outside the body for a time sufficient to enable the larva to develop. The prevalence of this harmless parasite gives, I imagine, an excellent minimal value for the excremental contamination of the intimate surroundings of a population. I have found, for instance, that three-quarters of the men who work in the abominable filth of some of the Cornish mines, are infected with *Trichocephalus;* and even higher percentages have been constantly found to accompany the elementary habits of the natives of the East.

If, then, fœcal contamination were the main factor in the occurrence of ankylostomiasis, the disease would be prevalent all the world over. The second condition is, however, the one which determines the geographical and topical

incidence. Epidemiological observation confirms the suspicion which is aroused by laboratory experimentation—that *Ankylostoma* would be found to be prevalent only where the eggs, on leaving the host, find those conditions of temperature, moisture, etc., which enable the larva to develop to the infective stage. Generally speaking, the geographical distribution of *Ankylostoma* is a question of temperature. It has been found in almost all parts of the tropical and sub-tropical world, and in all probability a short search would soon reveal its presence in those districts from which it has not yet been reported. In Europe it occurs among the general population south of the Alps; it is very common in northern Italy, and occurs in Hungary. It is probably common enough in Spain, though there are no details available. In northern Europe it has been found in those areas in which the necessary constant warmth has been provided by artificial means; the coal mines in Westphalia, in northern France, and in Belgium, and the metalliferous mines in Cornwall, have all suffered more or less severely from the ravages of the worm. The temperature in the underground workings of most extensive mines is high enough, at least in parts, to enable the eggs to hatch and the larvæ to grow to the encapsuled stage. Taking the possible lower limit as 16 deg. Cent., and the optimum as 25 deg. Cent., as defined by the experiments mentioned above, we find a striking confirmation in the following Table, showing the numbers per thousand of men suffering from illness in the different mines of the Westphalian coal-field, the mines being grouped according to their temperatures:—

Temperature.	Number of Collieries.	Number of Men Employed Underground.	Cases of Anæmia per 1,000 Men Employed Underground.
Less than 20 deg. Cent. (68 deg. Fahr.)	151	104,637	0.5
20 deg. to 22 deg. Cent. (68 deg. to 72 deg. Fahr.)	45	43,710	2.5
22 deg. to 25 deg. Cent. (72 deg. to 77 deg. Fahr.)	33	39,836	11.7
Over 25 deg. Cent. (77 deg. Fahr.)	12	9,853	39.9

These figures show that illness does not occur to any extent unless the temperature is such that the conditions are distinctly favourable for the development of the worm. In the inquiry into the prevalence of *Ankylostoma* in the Belgian coal mines, attention was paid rather to the number of infected men than to the number of cases of illness; and it was found that, under conditions of temperature at which development is possible, though not encouraged, a considerable number of miners became infected. Thus in sixteen mines, with a temperature of 15 deg. to 20 deg. Cent., the percentage of infected men varied from 4 to 52, though the cases of actual illness were relatively few.

It is perhaps interesting to consider the possibility of *Ankylostoma* living on the surface in this country. The Greenwich Records for Fifty Years show that the mean daily temperature exceeds 60 deg. Fahr. on an average of 71 days in the year; the average number of days per annum on which the temperature rises above 70 deg. Fahr. is 77, and on nearly four days per annum does it fail to fall below 60 deg. Fahr. The average number of days on which the temperature falls to freezing point is 56, and on nearly five of these it remains continuously below zero for 24 hours. Though the Greenwich Records represent the temperature of one of the colder localities in England, it would follow that *Ankylostoma* could only live under the most adverse circumstances, and each winter season would probably put a stop to the infection of any surface locality. Experiment shows, however, that it is quite possible for infected fæces left in the open in London to give rise to encapsuled larvæ. On two occasions in the early part of July of last year (1904), I found fully-developed larvæ in infected stools which had been left in the open air, and out of the direct influence of the sun, in a yard at Guy's Hospital. Similar experiments made during the period November to June gave uniformly negative results.

The question of moisture plays a less prominent part in the geographical prevalence of the worm, since few localities fail to provide the small amount which is absolutely necessary. The melancholy history of the Westphalian pits illustrates, however, the importance of moisture in the degree of vigour which the infection may display. The following Table gives the number of cases of marked anæmia observed in the Westphalian colliers from 1896 to 1902:—

1896	...	107	1899	...	94	1901	...	1030
1897	...	113	1900	...	275	1902	...	1355
1898	—	99						

In 1900 a system of watering the roads was made compulsory, in order to prevent explosions of coal-dust. This desirable object has been, I believe, obtained, but only at the price of an epidemic of ankylostomiasis, which has cost the coal industry many thousands of pounds, and which is still a source of no inconsiderable trouble and danger.

The only practical application of the need of oxygen shown by the developing embryo which is of material importance is this—that fæces deposited in and covered by water are safely disposed of, since no development will then take place. The anærobic qualities of the adult larvæ and its power of flourishing in water, render its transport by moving water possible and easy; in this way, infection may be brought over a short distance from an area of high temperature to one where the eggs would not hatch. Such circumstances as would render such a possibility of practical importance are of frequent occurrence in mines. When in water and undisturbed, the larvæ seem to quickly sink to the bottom; and since the finer sediment with which they are mixed in the polluted mud or dirt where they develop settles more slowly than the larvæ themselves, water which is perfectly clear on naked eye examination is probably free from infection.

The relation of the larvæ to poisonous solutions may occasionally play a most important part in the occurrence of the disease. An excellent example of this is found in the history of the Levant mine, in Cornwall. The present workings of this mine are to a large extent under the sea; the ventilation is very imperfect, and the temperature very high, rising from 71 deg. Fahr. at the bottom of the downcast shaft to 90 deg. to 93 deg. Fahr. in large areas of the deeper workings. The levels, ladders, etc., are wet and muddy, and the fœcal contamination extensive. A considerable number of men have returned to work in this mine from infected districts abroad, and some men who were actually known to harbour *Ankylostoma* had worked here for a time. The conditions, in short, for the introduction and spread of the disease were at least as favourable as those prevailing in other Cornish mines which were severely infected. Yet no trace of *Ankylostoma* could be found. The reason for this evidently lay in the nature of the water which permeates the mine. Some of this flows in from the land side, and is ordinary spring water, but

most of it is derived from the sea by leakage through the roof into the upper levels. As a result, the average content in salt of seven samples of water from different parts of the mine was 2.23 per cent., an amount which, as has been shown above, is sufficient to destroy the newly-hatched larvæ. There is no doubt that this mine owes its freedom from *Ankylostoma* to the somewhat undesirable thinness of the roof, and the resulting salinity of the water.

It will now be apparent that our knowledge of the life-history of *Ankylostoma* is sufficiently accurate and sufficiently—though far from altogether—complete to afford us very clear and definite indications in the matters of prevention and treatment.

Prevention of ankylostomiasis is secured by any system of disposal of excreta which is in accord with Western notions of common cleanliness.

The treatment of ankylostomiasis may be either the treatment of the infected individual or of the infected place. The former is beyond the province of the present paper; but, as it in practice becomes an integral part of the treatment of places, brief mention of it is necessary. We have, fortunately, in thymol a drug which acts as an exceedingly efficient anthelmintic for *Ankylostoma*, if used in the proper way. The first necessity is that adequately large doses should be given: there is a rather prevalent feeling that thymol is an extremely dangerous drug, and a consequent tendency to "be on the safe side" in the doses which are given. Small doses are "on the safe side" for both the host and the parasite, and in consequence no good results. The second necessity is that treatment should be repeated as often as is necessary to kill all the worms; this can only be judged of by very careful microscopical examination of the stool for eggs after each dose. It is worse than useless to treat an infected individual without taking the trouble to gain an accurate knowledge of the result: one dose often fails to kill all the worms, though the patient's general health may be much improved. As a consequence further treatment is not insisted on, and the worm-carrier, efficiently isolated while seriously ill, is enabled to go abroad and spread the disease still further.

The treatment of infected places opens up a very large, and in many places still untried, field. The indications are clear enough, but in practice the feasibility of following them out is of necessity an important consideration. It is obviously necessary to prevent further contamination, on the one hand by arranging that all fæces shall be

disposed of in an adequate manner; on the other, by treating everyone who harbours the worm until eggs are no longer present in the stools. Re-infection of the locality is thus prevented. How to destroy the infection already present must depend on local circumstances. It is practically certain that the larvæ, long-lived as they are, must die out sooner or later, and, since no reproduction of individuals takes place outside the body, evacuation of the locality would in the end be efficacious. If this is impossible, treatment with poisonous solutions might, I am convinced, be undertaken in some instances (notably, of course, in mines) with some prospect of success. In the case of coal-mines, evacuation would result in the production of an anaerobic atmosphere in which, after some time, the larvæ would perish.

Where the infected area is circumscribed, as in mines or tunnels, prevention is easy. The success with which the worm has been excluded from the Simplon Tunnel workings is a great testimony to the vigilance of those responsible for the medical care of the workmen; it is also conclusive proof that such disasters as that at the St. Gothard twenty years earlier are quite preventible. The cure, however, of even a limited underground area is a matter of difficulty, as the Westphalian experience has shown. A system of frequent examination of all underground workers, followed by treatment of those found to be infected, has been combined with apparently efficient efforts to overcome the peculiar prejudice in the matter of defæcation which seems to be common to all miners. By this means the amount of actual illness has been reduced to *nil*. It is, however, disquieting to learn that all the efforts which have been made during the last four years have failed to altogether eradicate the infection. At one colliery, for instance, the percentage of infected men was 20 per cent. at the first round of examination and treatment, 13 per cent. at the second, 8 per cent. at the third, and 3.6 per cent. at the fourth. The figure remained, however, about the same at the fifth, sixth, and seventh examinations. This persistence is probably accounted for by the fact that the pit is still infected, though fresh contamination has been stopped. There are, also, a certain number of errors of diagnosis, since worms may be present in the intestine, and yet no ova may be found in the stools; and a few cases have been met with in which the most vigorous anthelmintic treatment has failed.

Artificial alterations in the temperature and moisture

are in all cases extremely difficult, and in most cases quite impracticable. They will also do no more than influence the development of the eggs. The same end is attained more simply by securing that there should be no eggs. In a few cases dryness may be aimed at as an auxiliary measure, and in still fewer a slight diminution of temperature may be obtained, which may help to turn the scale against the worm.

In the tropics, where the infected locality is co-extensive with the inhabited area, little can be done beyond treatment of cases of definite illness as they arise, and prevention of a certain amount of infection by the protection of bare feet and legs. It seems to be beyond hope that the natives should be induced to assist by disposing of their excreta in appointed and suitable places.

It would seem unnecessary to remark that the recognition of *Ankylostoma* as one of the most potent causes of illness and inefficiency in hot climates is the first step in the way of treatment. It has, however, always been characteristic of the disease that its true nature should long remain unsuspected. In Puerto Rico, for instance, about one-quarter of all deaths were certified as due to "anæmia," yet it remained for Ashford to point out, in 1900, that the real cause was *Ankylostoma*. In the same way, it was not till 1902 that Stiles showed that ankylostomiasis is extremely prevalent in the southern parts of the United States. Yet, again, the worm caused much illness among the Cornish miners from 1894 onwards; but the real cause of "Dolcoath anæmia" remained unsuspected, and necessarily untreated, until it was pointed out at the end of 1902 by Haldane.

NOTE.—Further information with full bibliographies may be found in—*Report upon the Prevalence and Geographic Distribution of Hookworm Disease in the United States*, by C. W. Stiles, Bull. 10, Hyg. Lab., U.S. Public Health and Mar.-Hosp. Service, Washington, 1903, and *L'Ankylostomiase*, by A. Calmette and M. Breton, Paris, 1905.

DISCUSSION ON DR. BOYCOTT'S PAPER.

SIR PATRICK MANSON: I have listened with great interest to this admirable Paper, which to my mind is one of the most lucid expositions of the subject of Ankylostomiasis in medical literature, at all events in England.

There are one or two gaps which I should like to see filled, especially in reference to the life-history of the parasite. Dr. Boycott takes no notice of the views of

Giles and other observers, who believe that the Ankylostoma is capable of reproducing outside the human body as well as inside the human body; that the larvæ under certain conditions can become sexually mature outside the body, and so multiply without ever entering the human body.

Another point, as regards the life-history, which I should like to have seen discussed at greater length, is with respect to Looss's recent very important discoveries as to the way in which infection occurs. I understand that Looss is of opinion (as the result of experiment, I believe) that the only way in which infection can occur is by the larvæ of the parasite in the first place penetrating the skin, entering the circulation, passing through the lungs, undergoing certain changes there, and then entering the æsophagus, and so passing into the stomach. He also says that if the Ankylostome larva is introduced directly into the stomach it is digested and so disposed of; he considers that the object which is probably served by the passage of the larva through the lungs and through the blood is to acquire a certain amount of resistive power, which enables it to withstand the action of the gastric juice, and so enables it to get along without being killed until it reaches its proper habitat, the small intestine. I know that it has been shown by experiment that infection can be produced by the artificial introduction of Ankylostoma into the stomach; but Looss explains this by saying that the infection is not produced by the larvæ that proceed directly to the stomach, but only by larvæ arrested on their way to the stomach, and which have penetrated the walls of the æsophagus in the same way that they penetrate the skin when they obtain access through that channel.

I should be glad to hear whether any attempts have been made to secure the passage of the Ankylostoma larvæ directly into the stomach, without their being given an opportunity of attacking the walls of the æsophagus: for example, by introducing larvæ in gelatine capsules, or in any other way which would prevent them from attacking the walls of the æsophagus before getting into the stomach.

Ankylostomiasis is an exceedingly common disease in the East, and is by no means confined to natives. In this country I have seen three or four cases of apparently pernicious anæmia which, by examination of the stools with a microscope, were found to be Ankylostomiasis. I would

strongly recommend anyone who is consulted by an anæmic from the Tropics not to neglect the examination of the stools with the microscope. I recollect very well one case of a patient discharged from a hospital as the subject of an incurable form of pernicious anæmia; his stools were found to be loaded with the ova of Ankylostoma, and a few doses of thymol cured his pernicious anæmia.

The importance of the Ankylostoma in the Tropics is difficult to over-rate. Dr. Thornhill, who made a very complete study of the distribution and prevalence of Ankylostomiasis in Ceylon, says that it is far more important to the Cingalee than cholera, both directly and indirectly. The aggregate mortality is infinitely greater than from a disease like cholera, so prevalent is the infection.

I am perfectly sure that if we were to make a systematic examination of the stools of Lascars from Tropical countries, we should find that in at least half of them Ankylostoma are present: not in sufficient numbers, perhaps, to produce anæmia, but still the Ankylostoma is present. Everywhere throughout the Tropics you will find this condition.

I should like to ask whether Dr. Boycott found Anguillula in the stools of the miners in Cornwall as well as Ankylostoma; because in Tropical countries, in a large proproportion of Ankylostoma patients, Anguillula is also present. It is not of great pathological importance, but from a biological point of view it is interesting.

May I also ask Dr. Boycott whether he has made any experiments with filix-mas in these cases: it is said by some to be more effective than thymol. I am myself satisfied with the efficiency of thymol, which is an invaluable drug in the management of intestinal parasites in the Tropics; it will often procure the discharge, not only of Ankylostoma, but of round-worms, tape-worms, and flukes of several descriptions. Sometimes, after a sufficient dose of thymol you will find a perfect museum of organisms in the stool. I had one patient in the hospital last year—a Lascar from India—in whose intestines and blood we found at least half a dozen different parasites. He had Ascaris lumbricoides, Tricocephalus dispar, Ankylostoma duodenale, Filaria nocturna, and Bilharzia; he was indeed a perfect museum of parasites; it was possible to give a different clinical lecture upon him every day in the week.

As to the practical management of Ankylostomiasis, I may mention that, some time ago, I had a conversation with the proprietor of a sugar estate in Trinidad, where Anky-

lostomiasis is extremely common. He was put to great expense in consequence of the constant sickening of his coolies from anæmia. The cause of this anæmia he did not know, but he remarked that the coolies working in certain fields were extremely liable to a peculiar skin eruption, known as "coolie-itch;" and that all these coolies were, sooner or later, invariably struck down by a pernicious anæmia. Undoubtedly these coolies working in these particular fields had become infected through their skins by the Ankylostoma larvæ. Now, my friend the planter was not aware of the pathology of this "coolie-itch;" but he argued that the coolies became infected somehow through their skins by the germ which produced the "coolie-itch;" he believed the germ to be a bacterium; and he thought that if he could prevent the bacterial infection he could prevent the subsequent anæmia, and so prevent loss to himself. He set to work to try and find out some antiseptic which might be efficient as a protection against this infection, and he bethought him of an experiment which he had seen made many years before in Germany. He was a dyspeptic, and used to pay a visit to a certain German spa every year, and there he had become intimate with some farmers interested in goose-rearing; these farmers were a long way from the market, and in order to get the geese to the market they had to travel over great distances. The roads were flinty and rough, and the result was that the geese arrived at the market with their feet in a very injured condition. To prevent this, the farmers devised a peculiar kind of stocking. They made the geese, before starting on their journey, walk through a shallow layer of tar and then through a layer of sand; and in that way they were provided with shoes and stockings, and in these shoes and stockings they made their journey without injury. My friend the Trinidad planter thought he would apply this idea to his coolies; so he made them, before going to the fields to work, walk through some Barbados tar and afterwards through sawdust. The experiment had an excellent result. There was no more "coolie-itch" and no more anæmia.

I do not know whether that idea is practicable in every circumstance of plantation life, but certainly in this particular instance it was an efficient protection against Ankylostoma.

DR. LEIPER: Mr. Chairman and Gentlemen: I do not know that I can with any confidence offer any criticisms upon the excellent Paper which we have heard this evening;

but I will take this opportunity of expressing my thanks to the Honorary Secretary for having given me, as a non-member, notice of this meeting, and inviting me to be present.

The subject is one of great practical importance in this country—greater even, I think, than Dr. Boycott has pointed out. One would gather, perhaps, from this Paper that the occurrence of this disease in the British Isles was confined to the southern parts; but in Scotland, from which country I have recently come, we have, during the last eighteen months, met with cases of Ankylostomiasis in Lanarkshire. I think it is within the last eighteen months that Professor Stockman has put on record a very marked case in the mines of Lanarkshire; and I heard at the time of one or two cases of a "pernicious" anæmia, which were considered to be cases of Ankylostomiasis. It was possible that we were then on the verge of a somewhat extensive epidemic of this disease in the mines of the west of Scotland; and it is well that the importance of this disease and its prevalence should be brought under the notice of medical practitioners of mining districts, by a carefully-prepared *resumé* such as we have listened to this evening.

I should like to ask one or two further questions in the main line on which Sir Patrick Manson has opened, with regard to the method of infection. I have been interested to hear that Dr. Boycott had shown experimentally the efficacy of sodium chloride in preventing the development of the larvæ; and would like to know whether he has made any experiment with regard to the action of hydrochloric acid of various strengths on the embryos of different degrees of development. That might have given us some light on the question as to whether fully-developed embryos were able to pass through the wall of the intestine, or whether it was possible that the infection by means of the œsophagus is the only way, apart from the skin infection, by means of which Ankylostomiasis can be produced.

Dr. Boycott mentions a point which I should like to call in question. He (Dr. Boycott) says that it has been clearly established that the anæmia is not due to the blood having been extracted by worms: one would be inclined to question whether that has been definitely established, and whether in the cases of Ankylostoma duodenale there has been shown to be an antiferment or some toxic substance produced from the worms, somewhat similar to that which one can produce from Ascaris and other intestinal parasites. Dr. Stockman distinctly states that in his opinion the anæmia was caused by the actual loss of blood owing to the habits of these creatures.

In view of the opinion of so distinguished a hæmatologist as Dr. Stockman, who has given so much attention in particular to the anæmic conditions, I should like to ask whether Dr. Boycott has any further information with which to support the allegation he has made.

Then he stated that it has been somewhat remarkable that in some cases you get the fæces full of eggs which do not produce larvæ to any extent. I do not think that is a point of so very great importance as perhaps Dr. Boycott has made out, as it seems to me to depend almost entirely on the relative frequency of male and female in the intestine.

I should like to thank Dr. Boycott for his Paper, from which I have learnt a great deal, and in which I have been greatly interested, as well as for the demonstrations he has given us.

PROFESSOR KENWOOD: Mr. President and Gentlemen, I do not know that I can add anything to this discussion. I came to listen and to learn; and I am afraid if I ask many more questions that Dr. Boycott will be quite overwhelmed by the number of replies he will have to make.

But there are just a few points on which I should like to ask Dr. Boycott for a little more information.

With regard to the resisting powers of Ankylostoma larvæ to disinfectants, he has given us some interesting information. I should like to know whether he can give us any facts with regard to the efficacy of milk of lime, because in the German reports which I have read (including the Blue Book on the Westphalian Colliery attacks) they seem to be depending entirely on that disinfectant; at any rate, milk of lime was used very extensively. As Dr. Boycott has made no reference to it, I should like to know whether he considers it efficient.

It seems to me to be a most interesting point, that natives in tropical climes should suffer from this disease, whereas it appears, so far, at any rate, as northern Europe is concerned, that it is almost essentially an underground disease in Europe. In Germany this remarkable fact has been observed: that the miners who contract the disease do not give it to their wives and children, or other members of the family who are employed above ground. That is the statement given by the British Consul, who seems to have collected facts very carefully in Germany. If that is so, it is a remarkable circumstance, which is perhaps sufficiently explained by the peculiar conditions of moisture and temperature in mines. The conditions in

mines are not the same as above ground; there you get considerable heat and moisture combined, and at the same time unusual facilities for infection through the mouth and through the skin. In the case of these particular Westphalian mines, carefully-collected facts seemed to demonstrate that no known cases of Ankylostomiasis had been recorded in Germany prior to 1896; but that at the end of 1900 they commenced the practice of wet spraying in order to lay the dust. The infection seems to have been introduced prior to that; doubtless, it had been introduced on many previous occasions, but probably the moisture produced in the mine was the determining factor which enabled the disease to spread as it has done. Dr. Boycott points out what a costly business this spraying has been, but I think there is something on the credit side. In the first place, it has laid the dust, and thus prevented a great deal of respiratory disease; it has also proved entirely efficient in preventing explosions.

Lastly, is there anything at all in the horse theory which we read about a year or two back? Of course, one takes it for granted there cannot be. There was a very strong feeling in Germany that the appearance of this disease was coincident with the introduction of horses into the mines; and that feeling was held very generally by the medical men as well as by the miners themselves.

All the recommendations pointed out by Dr. Boycott are on sound scientific lines; but to get a miner to use sanitary boxes and wash his hands before eating, and so on, is, I am informed, an absolute impossibility in this country, if not in others. In Germany, they had tremendous difficulty in getting the men to take any precautions at all. I have been told that it would be absolutely impossible to get such regulations observed as would do away with all risk; therefore it seems to be very necessary indeed that men who are taken on in mines, if they do not belong to the district, should have their previous history thoroughly gone into, and their fæces carefully examined, before they are allowed to join the workers, having regard to the great difficulty there is in exterminating infection when once introduced.

Dr. Low: Sir Patrick Manson has raised one very interesting point, not mentioned by Dr. Boycott: the rhabditic or sexual stage of the Ankylostome outside the body, observed and described by Giles and others. This, I think, is an important fact in the life-history; and I remember Dr. Ozzard, when I was in British Guiana, being very

emphatic about such a thing taking place. He afterwards published an account of his experiments in the *British Guiana Annual.*

As regards Looss' experiments on the Ankylostomes entering the body through the the skin, and then going to the lungs, it is difficult to understand—still more so to prove—how the embryos crawl up from the lung through the trachea, and then down the œsophagus. That the embryo enters through the skin and gets to the duodenum is undoubted; but more work seems to be required to enable us to know how it gets there.

One point Dr. Boycott did not mention was the preesence of the Ankylostomes in the duodenum; it occurs there as well as in the jejunum. Another fact noted by Dr. Boycott, and that a very important one, was the presence of parasites in a host, without the production of any definite symptoms. We know, for instance, in filarial countries, that large number of the inhabitants have this parasite, and yet show no signs of the disease. While in Uganda studying sleeping sickness, I noticed this very markedly. In reports on the diseases of this country I had read that Ankylostomes were said not to exist; yet at the first autopsy I made I found fifty, as well as other intestinal worms. Further examinations of sleeping sickness cases and others showed that, though no symptoms of helminthological invasion might be present clinically, yet those individuals often harboured Ankylostomes, Trichocephalus, Ascaris lumbricoides, Filaria perstans, and Bilharzia. Though anæmia was present in some, it was not of the Ankylostomiasis type, and could always be explained as due to sleeping sickness and malarial complications. Again, the blood of many of those natives, though infested with so many worms, did not show any increase in the eosinophile leucocytes. This might be due to a gradually-acquired immunity, of course; but it illustrates very forcibly the danger of laying too much stress on this feature in the diagnosis of helminthiasis.

As regards the prevention of the disease amongst natives by sanitary measures, this would be very difficult. It would, I think, be impossible to get them to wash their hands or feet in tar, though in coolie gangs employed on plantations this might be accomplished by careful supervision. In such situations also much good might be done by introducing a system of proper latrines. Such methods are, I fear, however, impracticable for the African negro.

THE PRESIDENT: I will now ask Dr. Boycott to reply; but before doing so, there are one or two points to which I should like to draw attention.

We are again singularly fortunate in our Paper, and the Paper we have heard to-night is a worthy and fitting sequel to the one we listened to last month.

All those who have hitherto spoken are authorities on the subject; indeed, we are very fortunate in having those present who can speak from personal knowlege on this subject. I cannot myself speak from personal knowledge, but there are just one or two points that I wish to comment upon.

The first is the possibility of this disease taking root in this country. We have heard something of it in the Cornish mines, and of the absence of it in the West Riding coal-mines; but the fact that it is known in the Lanarkshire mines is news to me at least. It may be that the disease has a wider extension than we suppose at present; indeed, the question is full of possibilities, but the one that appeals to me particularly is the possibility of its existence in factories and workshops. We have been told that the reason of its being found in mines is the artificial climate to be found there. I am sorry to say that an artificial climate of that kind is not entirely limited to mines; we have artificial climates just as hot and just as humid in many factories. One is reassured to learn from Dr. Boycott's Paper that that is not enough; and that if the sanitary arrangements are of a kind which can be tolerated in modern civilisation, the danger is practically small. That is certainly reassuring; but during the last twelve months I have had something to do with organising a crusade in that matter, and I am afraid I must admit that there are factories and workshops in which the conditions in regard to latrines are as primitive as can be found in any human habitation, and in some cases are absolutely wanting. The particular point is, that in some of these hot and humid work-rooms there are latrines in which the conditions, so far as temperature and humidity go, would not be unfavourable to this disease. Still, we have never heard of Ankylostomiasis in connection with factories and workshops, and I hope we shall never do so. We shall take the lesson to heart, and it will afford an additional reason for pressing for this sanitary accommodation, which is so absolutely necessary from other points of view.

I quite agree with what has been said as to the importance in any epidemic of the under-current, so to speak, of slight and often unrecognised cases. There seem to be

always, besides the severe cases, a considerable number of slight but still quite obvious cases. In the case of Ankylostomiasis these cases may be unattended by bad symptoms, and sometimes even unattended with the discharge of ova, which alone would enable us to recognise the disease. This point has been worked out in this Paper and in others, and I personally attach great importance to it: that there may be a wider under-current than we suspect of cases which are not, in the clinical sense, cases at all.

It now only remains for me to tender to Dr. Boycott our cordial thanks for his most admirable Paper, the value of which is greatly increased by the illustrations and demonstrations he has given us, and by the singularly happy way in which he delivered his address.

Dr. Boycott: I have spoken with some diffidence on this matter altogether in the presence of those who, like Sir Patrick Manson and others, know so much more about the matter than I do.

With regard to developing outside the body, I had wanted to say something about that, but I feared to make my Paper too long. Personally, I am thoroughly convinced that there is no sexual development outside the body, and for two reasons:—

1. I have entirely failed to observe such a development myself; and I have taken a great deal of pains to try to notice such a development by feeding larvæ on extracts of intestine and blood, and by keeping them in anærobic conditions, and all the rest of it.

2. My attention has been strongly drawn to the fallacy that underlies all these experiments outside the body, and that is this: all over the world, and in practically every soil, are nematode worms, which, so far as their larvæ are concerned, are practically indistinguishable from Ankylostoma. If you keep these earth nematodes warm, in a few days they develop into sexual forms, which are exactly the forms which Giles originally described and figured, and they have the ordinary sexual apparatus which is common to nematodes. What people imagine to be a sexual stage of Ankylostoma is really an earth nematode. It is remarkable how many of them there are which are practically indistinguishable from Ankylostoma. Mr. Cadman, who was lately an Inspector of Mines in Staffordshire, took a great interest in Ankylostomiasis, and went about with me a good deal in connection with this matter; he was extremely helpful in collecting specimens, and so forth. I once received some specimens from him which he had obtained

in Shropshire, with a communication stating that he had found fæces underground which contained Ankylostoma eggs. On making an examination I found that they did contain eggs which resembled Ankylostoma, but which were really the eggs of a nematode; there was no suspicion that these mines had been infected with Ankylostoma. The fact is, there are worms which are doing their best to pretend they are Ankylostoma, and so mislead people. Other observers have also been misled by them. The worms which have been described in some Papers in the last three numbers of the Royal Army Medical Corps' *Journal*, are clearly ordinary earth nematodes. Major Smith concludes that they do not represent the sexual stage of Ankylostoma.

With regard to the experiments of Looss as to the mode of transmission from the skin to the intestine, Dr. Low mentioned that they are not altogether satisfactory. As to thinking that infection cannot take place through the mouth, I do not believe it for a moment. The experiments of Leichtenstein are quite clear, and all my own experience makes me think that mouth infection is quite possible. I made a large number of attempts with developed larvæ, hatched artificially, to infect myself through the skin: with the result that there was no trace of infection, either in my blood or in my stools; afterwards I got the infection immediately through my mouth; so that I have no doubt that it is quite possible to become definitely infected through the mouth.

It is hardly credible that the larvæ could penetrate the walls of the œsophagus, and my own experiments did not at all bear out the idea that the larvæ are very quickly digested in the stomach. I found that the fully-developed larvæ would live for two or three days in artificial gastric juice.

I am very grateful to Sir Patrick Manson for the idea of gelatine capsules. I have almost got some excellent volunteers for experiments, and I will certainly try that one if I succeed in getting them.

With reference to the presence of Anguillula in the Cornish cases, it was present—or at any rate a worm very like it: it seems to be almost inseparable from Ankylostoma. It has also been found in the Westphalian cases, and in the north of France.

My experience with thymol is the same as that of Sir Patrick Manson. Once we did experiment with other drugs. We gave one man (before the disease was diagnosed) two drams of filix-mas, and we did not get a single

worm: the same man afterwards had a dose of thymol, and we got five hundred worms in his stools. Needless to say we did not rely any more on Felix-mas. I have heard that β naphthol is more efficacious than thymol.

Dr. Leiper mentioned the cases in the Lanarkshire mines. I do not understand whether they were cases actually infected in Lanarkshire, or brought there from abroad.

DR. LEIPER: There was no record of these miners having been abroad.

DR. BOYCOTT: That is very unfortunate and disquieting, because it has always been assumed that the mines in Lanarkshire are too cold for Ankylostoma to develop in: the temperature, as a rule, is not much above 60 deg. Fahr. It only illustrates the danger that the coal industry undoubtedly runs from it.

With regard to anæmia being due to bleeding, in the first place it seems to me extremely doubtful whether the worms do suck very much blood. If you examine fresh worms, the vast majority have no appearance of having blood in them. In the second place, the observations would seem to be absolutely conclusive against the anæmia being due to this cause. I refer to the observations carried out by Haldane. These observations go to show that the total hæmoglobin in the blood was very little less than in normal people. Now, in Ankylostoma the total volume of the blood is very much increased, and it is increased practically in proportion to the diminution of the percentage amount of hæmoglobin; that is, if a person has 50 per cent. of hæmoglobin in his blood, he has his blood volume doubled, so that the total hæmoglobin in his body is the same. The anæmia is produced by dilution of the blood, and not by any deficiency of the total hæmoglobin in the whole body. I am quite aware that the acceptance of this conclusion depends upon whether you believe the method of determining the total amount of hæmoglobin in the body to be sound. I believe it is a sound method, and I am quite satisfied with it. I would also remind Dr. Leiper that Dr. Stockman used to believe that pernicious anæmia was due to repeated small losses of blood.

I have not made any experiments with regard to milk of lime, although it has been tried in Germany.

The idea of the infection passing through horses must be wrong. Horses have an Ankylostoma, but it is one which is special to themselves, and cannot infect man, and *vice versâ*.

Professor Kenwood has referred to a practical point—as

to the possibility of getting miners to use a sanitary pail. Any coal-owner who has not tried it will tell you that it is absolutely impossible; but I know of a mine where, for the last three or four years, sanitary boxes have been in use, and there is no trouble with the men whatever. It is a large mine, and the total cost of the thing has turned out to be practically nothing. With a mine in which about nine hundred men are working, it has cost about £1 a week, all told. I believe that if the matter is tackled properly, there would not be the slightest difficulty in getting it done. Even in Cornwall the men have taken to it (comparatively!) quite kindly, and Cornishmen are notorious all over the world for their habits in this respect.

I was interested in what Dr. Low said as to the absence of Eosinophilia in natives, and I was wondering whether it might be due to the fact that they had had parasites all their lives. I should like to ask him whether he found it in children at all?

DR. LOW: Yes, I did find it in children.

DR. BOYCOTT: That might explain it, perhaps.

The idea suggested by the President, that it is possible for ankylostomiasis to be spread in factories is a new one to me; and one, I trust, that will not be justified by experience.

As to the mines, of course there are a large number of mines in England which are eminently suitable places; and no doubt, unless they do take some care in the matter, some time or other they are bound to get it, considering how common it is all over the Continent. Now that things are "looking up" in the mining world, undoubtedly they will have a good many people—under-managers, for instance—now working in Continental mines, returning to England, who may bring infection with them. Once Ankylostoma obtains a firm footing in a mine, getting it out again is hardly compatible with going on working the mine, and it does amount to being a very serious matter.

As to the cases which may have no ova in their fæces, everyone has seen cases in which, from time to time, no ova could be found, but in which the ova were occasionally present; and other cases in which no ova were found during life, but the worms were found in the post-mortem afterwards, so that it was quite clear that they had been infected.

ON THE SPREAD OF SMALL-POX OCCASIONED BY SMALL-POX HOSPITALS DURING THE EPIDEMIC PERIOD, 1900 TO 1904;

AND

ITS RELATION TO ATMOSPHERIC CONVECTION.

By G. S. BUCHANAN, M.D.

(*Read: Friday, April 28th*, 1903.)

In 1894 the subject of the spread of small-pox occasioned by Small-pox Hospitals, in its general aspects, was brought before this Society in the admirable paper by Dr. J. C. McVail, which appears in the Thirteenth Volume of our *Transactions*. For more than six years afterwards there was practically no opportunity of adding to our knowledge of the matter, as during this time the country was almost free from small-pox. Now, after the usual fashion, a period of small-pox prevalence has succeeded; and at one time or another during the last four years—1900-01, 1901-02, 1902-03, and 1903-04—epidemics of considerable magnitude have occurred in London and in various parts of the kingdom.

The object of this paper is to inquire whether the ability of small-pox hospitals to cause the spread of small-pox in their vicinity has been again demonstrated during this epidemic period, and to consider whether the later facts which have been ascertained on this subject support or are opposed to the explanation of this circumstance by the assumption that particulate infectious matter from a hospital containing acute cases of small-pox is on occasion conveyed through the atmosphere in such a way as to infect susceptible people at considerable distances from the hospital.

During the four years in question, the number of hospitals available for the isolation of small-pox has increased all over the country. It may be said, indeed, with an approach to truth, that during this period every case of small-pox in the kingdom which has been detected has been isolated in a hospital of some kind. The exceptions to this statement, though themselves numerous, are re-

latively insignificant. At first sight, therefore, we should expect a superabundance of material to be available for our proposed inquiry. But it is, of course, otherwise. The administrative lessons of former epidemics have nearly always been taken into account by the medical advisers of sanitary authorities, in determining whether the use of particular hospitals for acute small-pox cases should be permitted, or in selecting the site on which a new small-pox hospital is to be erected or on which temporary accommodation for small-pox is to be improvised. For example, the use for acute cases of small-pox of a hospital which has a large population living within half a mile of it has been altogether exceptional. In the majority of instances it is useless to inquire whether a given hospital has spread small-pox, after the fashion of town hospitals in former years, among those who dwell in its neighbourhood or otherwise are habitually brought into its vicinity. The number of such persons is too small; the most minute inquiry as to the incidence of small-pox amongst them could not, in the nature of things, bring out data sufficient to enable inferences to be drawn with any degree of certainty.

We must turn, therefore, to special cases. I propose to refer to the experiences of London, Glasgow, Liverpool, Manchester, and Gateshead. Each of these five great towns has suffered from epidemic small-pox in the course of the last four years; and for each the incidence of small-pox on locality in relation to the small-pox hospital or hospitals at which the patients were isolated has been carefully studied. Fortunately for the brevity of this paper, the results in each instance are published and recorded in considerable detail, so that I need only refer to the principal facts of each.*

* *London: Hospital Ships in Long Reach.*—Dr. J. C. Thresh, Trans. Epidem. Soc., 1901-2. Dr. G. S. Buchanan, Report to Local Government Board on Small-Pox in the Union of Orsett, 1901-2.

Glasgow: Belvedere Hospital.—Dr. A. K. Chalmers, Report to Corporation of Glasgow on Small-Pox, 1900-1902.

Liverpool: Three Hospitals.—Dr. R. J. Reece, Report to Local Government Board on Small-Pox and Small-Pox Hospitals in Liverpool, 1902-1903.

Manchester: Clayton Vale Hospital.—Dr. J. Niven, Annual Reports of Medical Officer of Health for 1902 and 1903.

Gateshead: Sheriff Hill Hospital.—Dr. G. S. Buchanan, Report to L. G. B. on Small-Pox in Gateshead and Felling, 1903-1904, in Relation to Sheriff Hill Hospital.

N.B.—Each of these Reports contains detailed maps of the districts in question.

LONDON.

Metropolitan Asylums Board Hospital Ships in Long Reach.

It is natural to begin with London, where formerly small-pox hospitals played so prominent a part in determining the local distribution of small-pox. A brief reference to this earlier experience is desirable, in view of what follows. It will be remembered that the Metropolitan Asylums Board began to isolate small-pox during the great pandemic of 1871, and that until 1886 the hospitals used for the purpose were situated within or on the ontskirts of the Metropolis, being in each instance surrounded by, or at the edge of, thickly-populated areas : *e.g.*, the hospital at Fulham (the present " Western "); at Stockwell, ("South-Western"); at Deptford, ("South-Eastern"); and at Hampstead, ("North-Western"). It was in respect of Fulham Hospital, during the epidemic of 1881, that Mr. W. H. Power demonstrated the influence exercised by the hospital on the prevalence of small-pox in its neighbourhood. Mr. Power showed that, following on the use of this hospital for small-pox, a graduated intensity of small-pox incidence upon the surrounding houses was observed, the percentage of houses invaded by small-pox in the neighbourhood of the hospital becoming gradually smaller as the distance of these houses from the hospital increased. The influence of the hospital in this way was most marked within half a mile. It was manifested, however, in the zone lying between half and one mile from the hospital. This graduated incidence from centre to periphery was not to be explained by the hospital operations, and it did not follow lines of communication and traffic to and from the hospital.

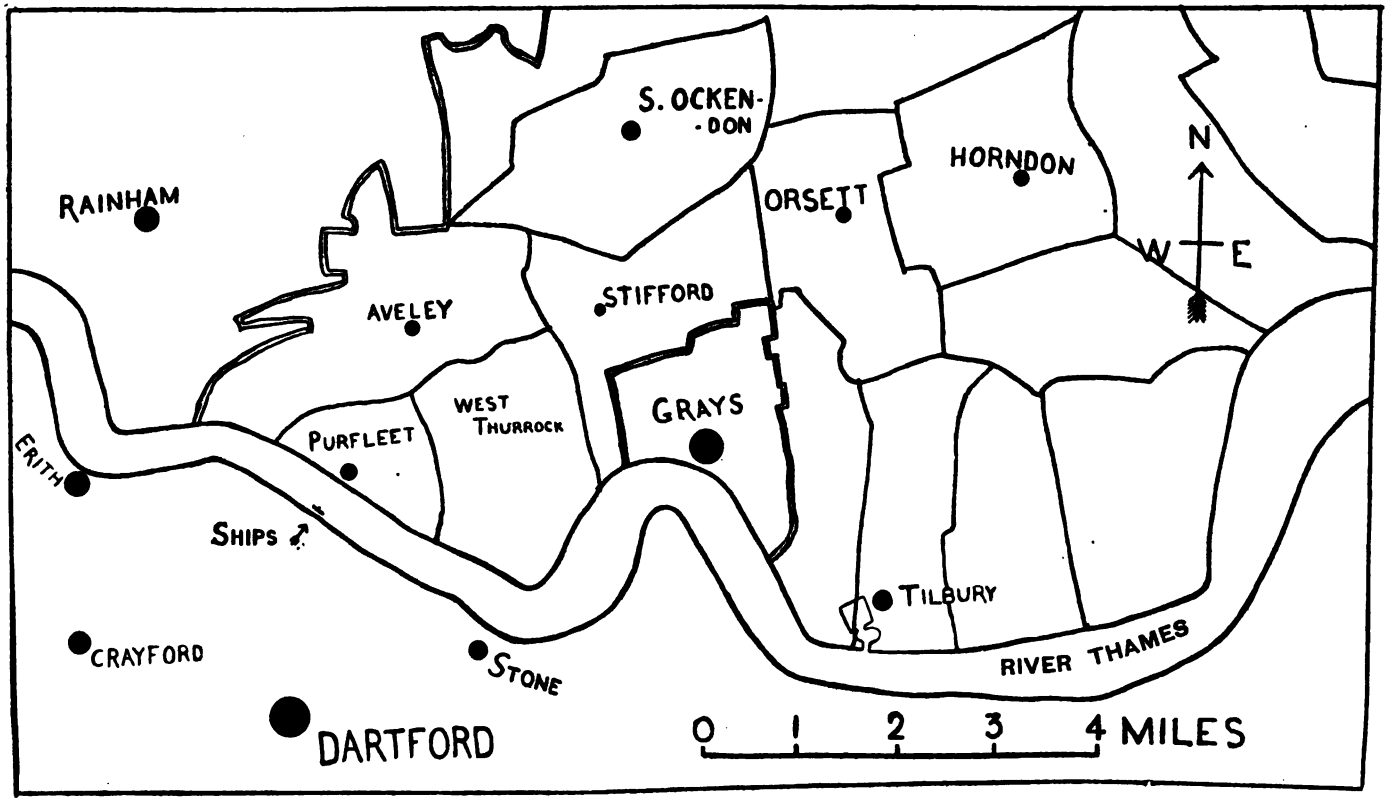
The striking facts obtained with regard to the Fulham Hospital in 1881, which formed the basis of the theory of aerial convection of small-pox formulated by Mr. Power, inevitably led to inquiry whether other London small-pox hospitals had caused spread of the disease in their neighbourhood; and whether, or to what extent, such spread could be prevented by stringent administrative regulations and precautions. Answer to the first question was furnished by Mr. Power's " Statistics of Small-pox incidence in the Registration Districts of London, relatively to the operation of Small-pox Hospitals in the Metropolis," published in the Report of the Medical Officer of the Local Government Board for 1886—a series of chapters dealing with local incidence in the Metropolis for 1876-1885. This

elaborate investigation made it clear that, for whatever reason, the local distribution and intensity of small-pox in the Metropolis during the epidemics included in the period under review, had been determined to a very marked extent by the operations of the London hospitals receiving acute small-pox cases. In particular it brought out instances in which the opening for small-pox of a hospital in a part of London hitherto comparatively free from the disease, (*e.g.*, Hampstead, in 1884), had been speedily followed by a severe local outbreak; and in general, the facts afford strong evidence that the persistence of small-pox in London, and the excessive prevalence of the disease in the Metropolis relatively to that in the provinces during this period, were to a large extent attributable to the use for small-pox of the London hospitals of the Metropolitan Asylums Board.

Opportunity of investigating the second question meanwhile had come during the epidemic of 1884-1885. The incidence of small-pox in the neighbourhood of Fulham Hospital in that year was again studied with great care and in great detail by Mr. Power. Notwithstanding a great variety of special and stringent precautions adopted at this hospital during this period, with a view to prevent opportunities of infection arising through hospital communications and traffic, the experience of 1881 was repeated. Again, there was exceptional incidence of the disease on dwellings near to the hospital, manifested within a few weeks of the admission of acute cases thereto, and again the characteristic graduation was observed.

Repeated demonstrations, therefore, of the mischief occasioned by treating acute small-pox cases in London hospitals left the Metropolitan Asylums Board no option. Henceforth they took all cases of this disease to hospitals outside the Metropolis altogether—namely, to the Hospital Ships in Long Reach, in connection with which was the Gore Farm "convalescent" small-pox hospital at Darenth.

I come now to the experience of London during the epidemic of 1901-2. This epidemic began in August, 1901. During the first fortnight in September the number of cases brought to the Hospital Ships was 75; in succeeding fortnights the corresponding numbers were 74, 71, 89, 211, and 240. Between November, 1901, and June, 1902, the average daily number of patients under treatment at the ships was 164, the maximum being 271 and the minimum 97. The reason that these figures were not even larger was that the bulk of the milder cases were sent on


S. OCKEN-
-DON
RAINHAM
ORSETT
HORNDON
N
W
E
AVELEY
STIFFORD
WEST
THURROCK
GRAYS
PURFLEET
ERITH
SHIPS
TILBURY
CRAYFORD
STONE
RIVER THAMES
DARTFORD
0 1 2 3 4 MILES

to the Gore Farm Hospital within a few days of their arrival at the ships. This circumstance should be noted: it entailed a concentration at the Hospital Ships of a large number of cases in the acute and most infective stage of the disease, together with cases which were too severe to allow their removal. The concentration and amount of infective material, be it observed, was far greater in the Hospital Ships than in any of the provincial hospitals to which I refer below.

Consider next the incidence of small-pox from September, 1901, to June, 1902, in the neighbourhood of these Hospital Ships: (1) on the Essex shore, which at its nearest part is separated from the ships by almost the whole breadth of the Thames, here nearly half a mile across; (2) on the Kent shore, off which the ships were moored; 3) on the river itself.

Many of the facts regarding the Essex shore were brought before this Society early in 1902 by Dr. Thresh; others were published in my Report to the Local Government Board on Small-pox in the Orsett Union, issued at the end of that year. The large area to be considered is indicated on the rough map annexed. It comprises twelve parishes in the Rural District of Orsett, and also the Urban District of Grays, a riverside town three miles below the ships. Scattered through the rural parishes are various collections of dwellings—some agricultural villages; others (*e.g.*, Purfleet and "West Thurrock") occupied by men employed at riverside works. This area, taken as a whole, suffered in 1901-2 from an outbreak of small-pox of remarkable severity. Per 1,000 of population, the attack rate in the twelve rural parishes was 18.5; in Grays it was 16. The attack rate in London during the same period was less than 2 per 1,000. In Holborn, the London borough most severely attacked, the attack rate was only 7.2 per 1,000. During the epidemic in London, small-pox appeared from time to time in other localities at similar distances from the Metropolis, but no epidemic comparable in severity to that in the Orsett Union occurred in any of these localities. A special reason for the exceptional severity in the Orsett Union had, therefore, to be found. It might be that the districts concerned were badly organised to deal with small-pox: that the epidemic found them without means of isolating cases of the disease, and without adequate means of disinfection. Or—as many of the children were known to be unvaccinated—it might be that infection had been spread wholesale by means of

schools. A large section of the population might have been refusing vaccination and re-vaccination, and the magnitude of the epidemic might, consequently have been due to excessive small-pox among "contacts." Or, again, the organisation of the sanitary departments concerned might have broken down, with the result that cases were not promptly detected and dealt with. Answers in considerable detail to these and other conjectures which naturally were forced upon anyone undertaking a systematic inquiry into the outbreak, will be found in my Report. From the first, there was no failure in provision of hospital isolation for small-pox cases. Throughout the epidemic, notified cases of small-pox were promptly removed to hospital. Disinfection and other preventive measures adopted by the sanitary authorities concerned, were, upon the whole, efficiently carried out. Each district had the services of an excellent and hard-working sanitary staff. Excessive prevalence of small-pox among "contacts" was not a conspicuous feature of the epidemic, and in at least four-fifths of the dwellings invaded by small-pox, no case of the disease occurred among the "contacts." The occurrence of small-pox among unvaccinated children had been frequent, and was responsible in material degree for the magnitude of the epidemic, but there had been no instance of wholesale spread of small-pox among children, such as occurred in Gloucester in 1895. After the first few months of the epidemic, the susceptibility of the general population to small-pox had been very greatly decreased, in consequence of the enormous amount of vaccination and re-vaccination offered and accepted in the Union.

It was, however, impossible to neglect the obvious key furnished by study of the local incidence of the disease. The place at which the epidemic had its beginning (in September and November, 1901) was the small community of Purfleet. Not merely to begin with, but throughout the whole epidemic, small-pox prevailed in Purfleet to a much greater extent than in other portions of the Union. The places which, next after Purfleet, were most heavily attacked were the portions of the Union nearest to Purfleet. In parishes between two and five miles from Purfleet, including Rainham, which is outside the Orsett Union, the introduction and recurrence of the disease were in many instances found to be attributable to infection contracted at Purfleet. This was also the case at Aveley, Grays, West Thurrock and South Ockendon. In each of these parishes the first cases of small-pox appeared to have contracted

their infection at Purfleet. To a less degree, the history of Purfleet in regard to small-pox was repeated during the latter part of the epidemic in the larger community of "West Thurrock,"* which lies between Purfleet and Grays, from two to three miles in direct line from the Hospital Ships. The facts and figures as to local incidence are given in detail in my Report.

What, apart from their proximity to the Hospital Ships, were the peculiar circumstances in which this collection of six hundred residents at Purfleet, together with another hundred or more people working at Purfleet and returning every day to dwellings within a few miles of it, came to suffer so severely? Apply to Purfleet the conjectures with which we approached the whole Union. The matter was carefully investigated by Dr. Corbet, Dr. Thresh, and myself. The comparative smallness of the population concerned here made the answers easier. And they were definite. Defects in isolation, in disinfection, or in contact vaccination; school infection; re-vaccination refusals; multiple cases in houses—none of these familiar factors in small-pox epidemics, and no combination of them, could be accepted as furnishing anything like a sufficient explanation of the recurrence of the disease week after week and month after month in this community. The local medical practitioner, the medical officer of health, the sanitary inspector, the inhabitants themselves—all were on the lookout for the occurrence of the initial symptoms of small-pox—I would dwell particularly on the promptness with which cases of small-pox in Purfleet were detected as they occurred, and on the rarity of "missed" cases there. Most persons in Purfleet were vaccinated or re-vaccinated during the early part of the epidemic, and the number of residents susceptible to small-pox by March was apparently reduced to something under forty, a number of these being persons newly arrived in the place. Nevertheless, cases continued to occur after March among these few remaining susceptibles.

And all this, as Dr. Thresh showed, has happened before, though on a smaller scale. Both in 1884-5, and in 1892-5, years in which numbers of small-pox cases were under treatment at the Hospital Ships, Orsett Union suffered from small-pox. For 1892-5 facts were available, and are given by Dr. Thresh and in my Report, to show that in each year corresponding to the use of the ships for small-

* As in my Report to the L. G. B., I have subdivided the large parish of West Thurrock into (1) Purfleet, and (2) "West Thurrock."

pox, Purfleet and West Thurrock suffered out of all proportion to the rest of the Union.

As to the Kent shore: I will pass over facts which I have recorded as to the first occurrence of small-pox in 1901 at Erith, and in parishes of the Dartford Rural District: places two miles or more from the ships; and deal solely with the uninhabited flats within a mile of them. On these flats, within a quarter of a mile of the ships, a large number of workmen were employed during and after December, 1901, in erecting a temporary hospital on shore, Up to February 27th, 1902, no case of small-pox was received into any shore buildings. From time to time during these three months, small-pox occurred among the workmen—at least 60 such cases were detected. Persona communication between members of the staff and these workmen cannot, however, be excluded as one of the causes of the small-pox among them.

As regards vessels ascertained to have cast anchor in Long Reach near to the Hospital Ships, instances are given in my Report, in which members of the crew of such vessels were found to have sickened with small-pox about twelve days afterwards, when in the ports of Rochester, Faversham, and London.

Revert for a moment to the outbreak on the Essex shore in the Orsett Union. The exceptional character of that outbreak demanded explanation, and one which would account for the wholly exceptional and sustained incidence of the disease on Purfleet and on places associated with Purfleet — the most conspicuous feature in the Orsett epidemic. Purfleet village and the riverside works there form the only population within a mile of the Hospital Ships; nearly all this population was within three-quarters of a mile. During the Orsett outbreak the administration of these ships entailed the concentration, almost by the hundred, of acute and severe cases of small-pox therein. These ships had been put there because hospitals in London receiving acute small-pox cases had been shown to spread small-pox in their neighbourhood: their influence in this respect having been traced on dwellings a mile from the hospital. In previous epidemics the operations of the Hospital Ships had coincided with prevalence of small-pox in Purfleet; during the present epidemic, crews of vessels moored in the river near the ships had become infected, while small-pox became rife among workmen on the opposite shore. All this affords strong grounds for inference that the exceptional severity of the epidemic in

Purfleet, (and, to a large extent also, directly or indirectly, the magnitude of the outbreak in the whole of the Orsett Union), stood to the operations of the Hospital Ships in the relation of effect to cause.

As regards *the provinces*, I will not attempt any retrospect of the various instances in which hospital influence has been traced in the case of provincial hospitals during small-pox epidemics in years antecedent to the four which we are now considering. A useful list of certain of these instances is given in Dr. Reece's Liverpool Report, to which I propose to refer later on.

GLASGOW, 1900-1904.—*Belvedere Hospital.*

We are fortunate in possessing a most instructive report by Dr. A. K. Chalmers, Medical Officer of Health for Glasgow, which deals in a variety of aspects and in well-arranged detail with the epidemic of small-pox in that city from 1900 to 1902. In dealing with the relative intensity of small-pox incidence during this period on various divisions of the city, Dr. Chalmers takes for his basis of comparison the number of cases which occurred in each division relatively to its population: no doubt for the reason that the tenement and flat system in Glasgow would introduce too many uncertainties if he attempted to make invaded dwellings his basis of reckoning. Dr. Chalmers divides 1900-1902 into pre-epidemic, epidemic, and recrudescent periods of small-pox, and he has studied each period fortnight by fortnight. The figures as to number and rates of attack in the seven large Divisions* of Glasgow are as follows:—

Small-pox in Divisions of Glasgow, April, 1900, to May, 1902.

Division.	Population.	Cases Reported in—		
		Pre-Epidemic Period, April to December.	Epidemic Period, January to July.	Recrudescent Period, November to May.
Central	111,784	83	139	48
East	173,104	216	863	205
North	166,825	26	100	103
South	132,718	41	200	65
West	61,092	23	29	24
South Suburban ...	64,205	7	48	9
North-West ...	51,984	1	10	15
Whole City ...	761,712	397	1389	469

* The proper name for what I have described as "Divisions" is "Administrative Districts." "Division," however, may save risk of confusion between these large "Administrative Districts" and the smaller "Sanitary Districts."

Attack Rate per 100,000 *of Population in*

Division.	Pre-Epidemic Period.	Epidemic Period.	Recrudescent Period.
Central	74	124	43
East	124	498	118
North	15	60	62
South	30	151	49
West	37	47	39
South Suburban	11	76	14
North West	2	19	29
Whole City	52	184	61

Even when the figures are thus massed together the disproportionate incidence on the East Division of the City during all three periods becomes evident. Belvedere Hospital, the sole institution used for the isolation of Glasgow small-pox cases, stands within this East Division, and on the eastern edge of it. This is shown in the rough map annexed. So much of the area within a mile of this hospital as is within the City of Glasgow lies wholly in the East Division. It contains a large population, viz., nearly the whole of the "7th Sanitary District" (population 66,197), and portions of the "8th" and "5th" Sanitary Districts. The population lying within half a mile of the hospital has not been separately estimated, but it may be noted that the area within half a mile is relatively less densely populated than that between half and one mile.

The massed figures just given, however, do not suffice to indicate the nature of the exceptionally heavy incidence of small-pox on the East Division. To appreciate this it is necessary to consult Dr. Chalmers' Tables of fortnightly incidence during the two years in question. These Tables show, as regards the pre-epidemic and epidemic periods, that small-pox was set going at the end of April, 1900, in the Central Division. During the first eight weeks, in which a total of 72 cases occurred, the disease became distributed throughout six of the seven Divisions of the city, without any specially noteworthy incidence on the East Division. After June 16th, however, the number of cases from the East Division began to be disproportionately large. The excess in this Division was maintained in almost every fortnight throughout the pre-epidemic period. It was augmented during the epidemic period which followed, from January to June, 1901. And, practically speaking, it continued until the epidemic ceased. After July, 1901, small-

N
W
E

pox disappeared from Glasgow; the hospital emptied. In November there was a recrudescence of the disease. This recrudescence occurred in the North Division of the city, two miles or more away from the hospital. After a period of eight weeks, other Divisions—the Central, South, and West—were affected, while a few cases also occurred in the East Division. At the end of twelve weeks, by which time 84 cases had occurred in the city and some 50 were in hospital, came an outburst in the East Division; and thereafter this Division continued to show an exceptional number of cases, fortnight by fortnight, until the end of the period of recrudescence. During this recrudescent period the proportion of susceptible persons in the whole city had become enormously reduced by the vaccination and re-vaccination performed in 1900 and 1901. It does not appear from Dr. Chalmers' Report that the proportion of inhabitants which had obtained protection during this time in the Eastern Division was any lower than in other divisions of the city.

Dr. Chalmers writes to me:—

"It should be made quite clear that in the Eastern Division of the City it was always easier to push re-vaccination than in other districts where public apprehension was less, so that the greater incidence of cases in the Eastern Division, especially in later stages of the epidemic period as well as during the recrudescence, and again during the return last year (1903-4), is all the more striking, because the excessive number of cases occurred, in reality, among a much smaller number of susceptible people than the relative populations would indicate."

In regard to the number of its vaccinated children at the beginning of the epidemic, the Eastern Division compared favourably with the rest of the city.

Dr. Chalmers tells me that he is about to publish further records of small-pox in Glasgow, which show, as I understand, that the experience of the recrudescence of 1902 has since been repeated. This time, small-pox again broke out in parts of the city remote from the hospital—away to the west. After an interval of several weeks, in which the hospital had again been taken into use, small-pox reappeared and persisted in the East Division; and in the result the East Division again contributed a larger proportion of cases than any other part of the city.

Now, as in Purfleet, all this had occurred before. Many years ago, during prevalence of small-pox in the early 'seventies, an aggregation of cases had occurred in the north of the city, around Parliamentary Road Hospital, which was then used for the isolation of small-pox. But from 1892 to 1897, years in which small-pox was present in Glasgow, and in which small-pox cases were admitted to

the Belvedere Hospital on the east, the proportion of cases contributed by the Eastern Division has been uniformly in excess of the proportion of population residing there. This Dr. Chalmers has shown by the following Table:—

Glasgow Small-pox.—Proportion from each Administrative District (or Division) of the Total Cases occurring in Several Years.

Year.	Total Cases.	Percentage of Total Cases.						
		East.	Central.	South.	North.	West.	South Sub-urban.	North-West.
1900-1	1,759	60.4	12.3	13.6	7.0	3.0	3.1	0.6
1892	78	28,2	19.2	26.9	3.9	20.5	1.3	—
1893	386	45.9	24.7	9.7	12.8	3.8	0.8	2.3
1894	49	32.7	36.7	16.3	3.2	2.0	4.1	—
1895	243	33.4	14.0	9.0	5.8	36.2	0.8	0.8
1896	5	—	—	—	—	—	—	—
1897	59	57.6	3.4	32.2	5.1	—	1.7	—
Percentage Population, 1901.		23	15	17	22	8	8	7

It should be added that for the pre-epidemic and epidemic period 1900-1901, Dr. Chalmers studied the incidence of small-pox not only on the seven Divisions of the city, but also on thirty-three smaller "sanitary districts" into which the city is also subdivided. The same result was again apparent. The attack rate of the whole city was 2·3 per thousand, that of the "7th Sanitary District," which includes most of Glasgow within a mile of the hospital, was 9.97 per thousand. None of the other thirty-two sanitary districts had an attack rate exceeding three per thousand, except the 8th Sanitary District, in which the rate was 6.46 per thousand, and the 11th, which had a rate of 4.2 per thousand. The 11th District was that in which the epidemic had begun. Part of the 8th District lies within one mile, and the whole of it within one and a-quarter miles from Belvedere Hospital.

Looking to all the circumstances of Glasgow, as set out by Dr. Chalmers, it is difficult to resist the conclusion that the operations of the hospital have influenced the distribution of small-pox in Glasgow to a very marked extent during recent epidemic years; and that the magnitude and persistence of the epidemics which have occurred during these years are to a large extent attributable directly and indirectly to hospital influence.

GATESHEAD AND FELLING, 1903-1904.

Sheriff Hill Hospital.

During 1903-1904 small-pox was prevalent, to a greater or less degree, in various towns at the mouth of the Tyne, including Newcastle, which itself contributed a considerable total of cases during these two years. The Borough of Gateshead, on the opposite side of the Tyne to Newcastle, sustained during this period what may be considered a pronounced epidemic (Gateshead, population 110,000; total cases, April, 1903, to May, 1904, 510; total dwellings invaded by small-pox, 316).

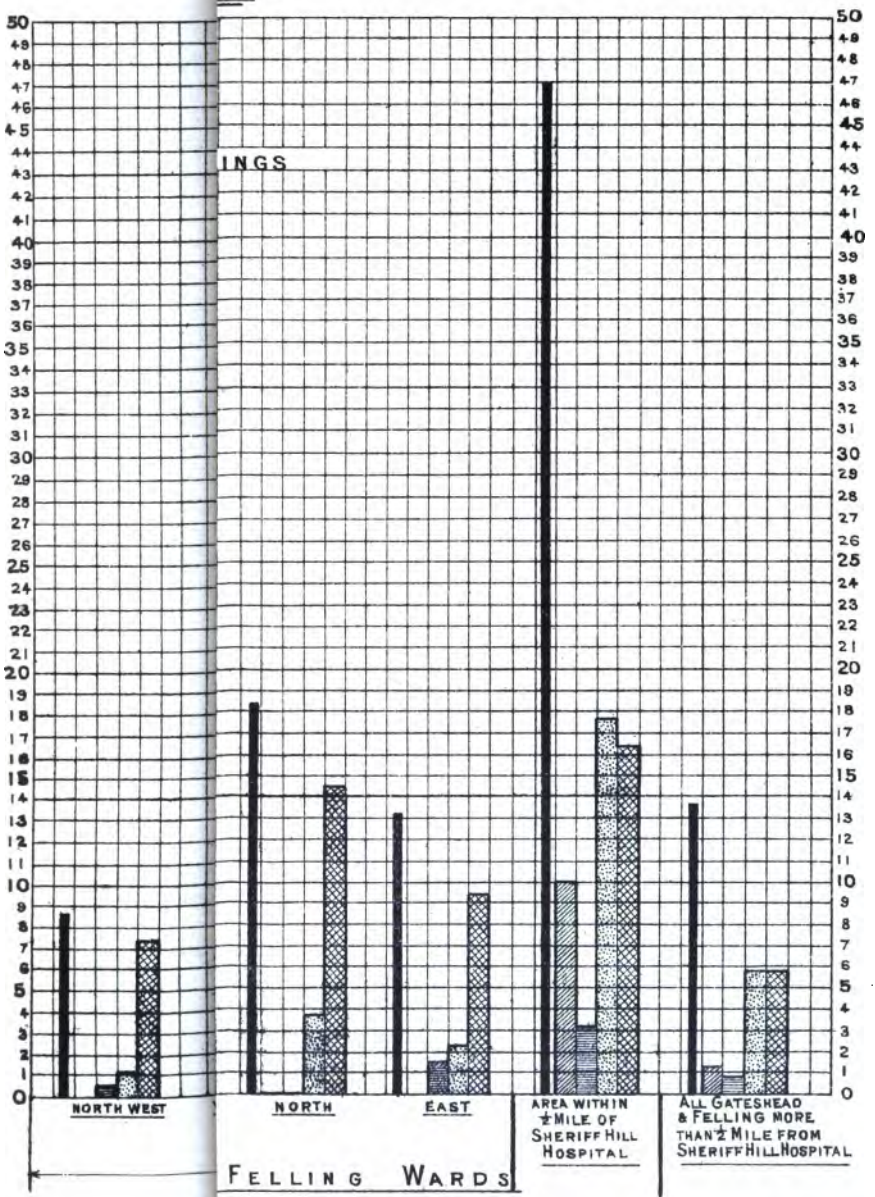
Of other boroughs and urban districts in Tyneside, that which showed the highest attack rate was the urban district of Felling, adjoining Gateshead, and to the east of it (Felling, population 23,000; total cases in the same period, 155; total dwellings invaded by small-pox, 106). The relative position of the two districts will be seen from the rough map exhibited. Early in 1904, Dr. Eustace Hill drew attention to the circumstance that the small-pox epidemic in Felling was mainly affecting, not its most populous part near the Tyne, but dwellings in the higher and comparatively open country to the south—in particular a village called Windy Nook, about two miles south of the Tyne, and close to the Gateshead - Felling boundary. The area specially affected was mainly that within a mile of Sheriff Hill Hospital, which was receiving all the Gateshead small-pox cases. The village of Windy Nook is between a quarter and half a mile from, and north-east of, this hospital. This, together with similar observations by Dr. Peacock, Medical Officer of Health of Felling, led to a detailed inquiry which occupied me for many weeks in 1904. into the local distribution of small-pox in both Gateshead and Felling. In Gateshead the distribution was not unlike that in Felling. Small-pox had been prevalent in various parts of Gateshead, at first (April to November, 1903), with comparatively slight intensity, later (December, 1903, to May, 1904), to a conspicuous and serious extent. But it was the more sparsely populated part of Gateshead—the higher and comparatively open ground near its southern boundary, comprising dwellings near Sheriff Hill Hospital—that had been affected to the greatest extent. By a special enumeration I ascertained that there were 1,297 dwellings (corresponding approximately to a population of 6,200) in Gateshead

and Felling, which are within half a mile of the hospital. The figures as to dwellings, together with fortnightly returns for each district, enabled a statistical comparison to be made between the incidence of small-pox on houses within half a mile of the hospital on the one hand, and on those in Gateshead and Felling, which are more than half a mile away on the other. The results were as follows:—

Dwellings newly invaded by small-pox within half a mile of the hospital	47 per thousand.
Dwellings newly invaded by small-pox more than half a mile from the hospital	13.7 ,,

I applied also a more searching test, by ascertaining the incidence of small-pox on the several *wards* of Gateshead and Felling. In this way it was possible to compare the incidence on the half-mile area with that on twelve other areas, each more than half a mile from the hospital, and each containing somewhere about the same population as the half-mile area. The result is shown in the diagram given in my report and here exhibited. The incidence on dwellings in the half-mile area (47 per thousand) was greater than the corresponding incidence in any ward of Gateshead or Felling more than half a mile from the hospital. The ward more than half a mile from the hospital which had the greatest incidence (25 per thousand) was the East Ward of Gateshead, many dwellings of which are within one mile of the hospital. The excess in the half mile area so conspicuously shown on comparison with the other twelve areas was not the result of a single special outbreak near the hospital during a limited time. It was found to exist in each of three periods of three months, and in the remaining period of four months and a half, into which the epidemic was divided; and on two or three occasions it was conspicuous within a few weeks of the hospital receiving an augmentation of acute cases. Moreover, the incidence on dwellings within a quarter of a mile of the hospital (82 per thousand) was greater than that on dwellings within the quarter to half-mile zone (35 per thousand), which again was greater than that on dwellings in the rest of Felling and Gateshead (13.7 per thousand). It should be added that the facts as to vaccination and re-vaccination gave no evidence of any special susceptibility of the population of the half-mile area as compared with the rest of Gateshead and Felling. As the epidemic progressed it was rather the other way—the half-mile area came to contain a larger proportion of insusceptible people.

-4.



8046.12.08

Weller & Graham Ltd. Litho. London.

These facts, together with study of fortnightly returns and maps showing invaded dwellings in the whole of Gateshead and Felling, indicated that this hospital exercised during 1903-1904 a very material effect in enhancing and maintaining the prevalence of small-pox in both districts.

MANCHESTER, 1902-1904.

Clayton Vale Hospital.

Dr. Niven has put on record the facts regarding the local incidence of small-pox in this city during 1902-03, and he has kindly furnished me with corresponding data in respect of 1903-04. From December, 1902, to June, 1903, there occurred in Manchester (population, roughly, 550,000) a total of 383 reported cases of small-pox; from June, 1903, to November, 1904, the total number of such cases was 219.

During these years the cases in question were isolated in Clayton Vale Hospital. The largest number of admissions to this hospital in any one fortnight in 1903 was about 60. Clayton Vale Hospital lies in the eastern outskirts of Manchester. The population in its neighbourhood is small. In 1900 it was ascertained that only 42 persons lived within a quarter of a mile of it, and 606 persons within half a mile. As regards the half-mile area, there are thus very few data to go upon in considering hospital influence: the conditions are very different from those of Gateshead. But I have been led to include Manchester in my list, for the reason that Clayton Vale Hospital has a considerable population resident between half and one mile from the hospital, namely, a large portion of the Newton Division (population 37,143), and nearly the whole of the Clayton Division (population, 9,795).

Dr. Niven's maps show that during 1902-03 only eight, and in 1903-04 only thirteen, cases of small-pox were reported to have occurred in dwellings situated between half a mile and a mile from this hospital. The cases among the small population resident within half a mile were only one and three respectively. Moreover, Dr. Niven notes that "a number of men were employed near the hospital laying drains, etc., but none of them developed small-pox, although most of them refused to be vaccinated."

So far, then, as conclusions as to hospital influence can be drawn from the behaviour of small-pox in inhabited

areas more than half a mile away from Clayton Vale Hospital during these years, and from the facts as to the small number of people living or working within one half-mile of the hospital, the results for Manchester are plainly negative. Contrast is at once suggested between this negative result and the positive result which was obtained in the case of inhabited areas more than half a mile from the Hospital Ships in Long Reach. To this I propose to revert later; but meanwhile it should be noted that Clayton Vale Hospital at no time during 1902-1904 contained any concentration of recent and severe cases of small-pox of the kind which were present on the Hospital Ships in 1901-02.

LIVERPOOL, 1902-03.

Priory Road, Park Hill, and Fazakerly Hospitals.

One of the most important contributions to the present subject is a report by Dr. R. J. Reece to the Local Government Board, issued within the last few weeks, which embodies the results of a laborious study of the question of Small-pox and Small-pox Hospitals in Liverpool, during the two years 1902-03. In this period, Liverpool (population, roughly, 720,000) suffered far more severely from small-pox than did Manchester. From December, 1901, to the end of 1902, the total number of cases reported (after deduction of certain cases brought by shipping) is given as 552. At the end of 1902 small-pox was increasing, and at the beginning of 1903 it assumed the proportions of a considerable epidemic, which continued until the end of June. 1,585 cases of small-pox were notified during the first half of 1903. Some 141 others occurred in the later months of 1903. Small-pox disappeared from Liverpool at the end of that year. During the whole period of two years, December, 1901, to December, 1903, the total reported cases are given as 2,278, and the total number of dwellings invaded as 1,632. The situation, as regards populous areas of two out of the three hospitals which were used for the isolation of small-pox during this period, was exceptional, and was favourable to the investigation of hospital influence. These hospitals are those at Priory Road and Park Hill.

Priory Road is a small hospital on the north-east of the city, with a total accommodation ordinarily reckoned as suitable for some forty small-pox patients. The maximum number of cases admitted thereto in any one fortnight of

the epidemic was sixty-five. This hospital has a considerable population living within half a mile of it, and a large population living within one mile.

Park Hill, a larger hospital than Priory Road, with a total accommodation of about 350 beds, is in the south of the city, about four miles from the Priory Road Hospital. The two hospitals are separated almost by the whole length of Liverpool. Park Hill also has a considerable population in its neighbourhood, the figures as regards houses being :—

—	Total Houses distant from the Hospital.			
	0 – ¼ Mile.	¼ – ½ Mile.	½ – ¾ Mile.	¾ – 1 Mile.
Priory Road Hospital ...	85	1970	7257	12412
Park Hill Hospital ...	171	2259	4563	5617
Fazakerly Hospital ...	2	166	867	1423

To these I have added the figures for the third hospital—Fazakerly—a large hospital outside the city boundary, which had accommodation for 160 small-pox patients. Fazakerly Hospital, unlike the other two, is in a comparatively sparsely-populated district. The situation of these Liverpool hospitals is shown on the rough map exhibited.

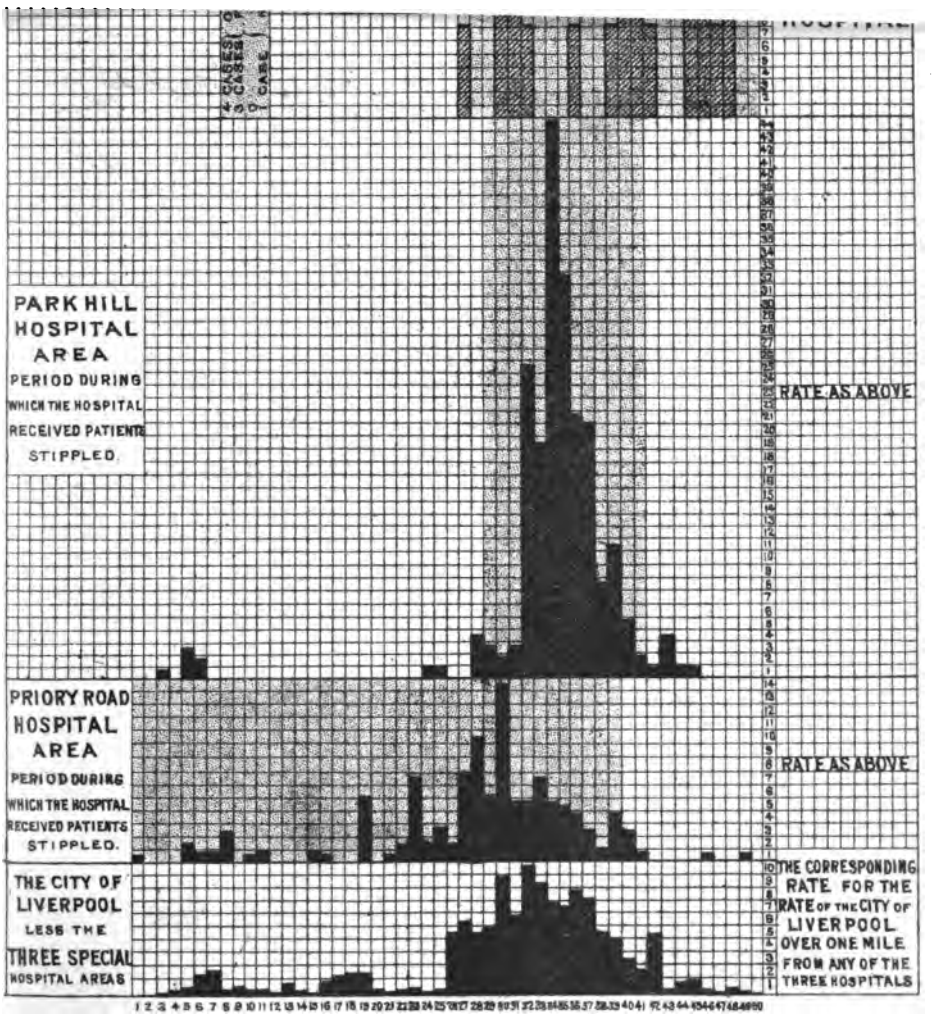
Liverpool did not have all three of its hospitals open for small-pox cases throughout the epidemic. The periods during which the several hospitals were open to receive cases were as follows :—

Period.	Approximate Number of Weeks in Period.	Hospital or Hospitals to which Cases of Small-pox were Admitted.
6th December, 1901, to 10th December, 1902	53	Priory Road.
10th December, 1902, to 12th January, 1903	5	Priory Road, Fazakerly.
12th January, 1903, to 26th May, 1903	19	Priory Road, Fazakerly, Park Hill.
26th May, 1903, to 1st July, 1903	5	Fazakerly, Park Hill.
1st July, 1903, to November, 1903	21	Fazakerly.

The extent to which cases of small-pox were admitted to the several hospitals during each of the above periods is shown by the diagram given in Dr. Reece's Report. The rapid admission of large numbers of small-pox cases into Park Hill Hospital in January, 1903, is well illustrated by this diagram.

Now, anyone knowing the facts only thus far, but acquainted with Mr. Power's researches on the behaviour of small-pox in London before 1885, would at once fasten upon this sudden opening of Park Hill Hospital in January, 1903, as the most obvious point for inquiry. What was the history as regards small-pox of the populous areas near this hospital before it was opened, and what happened afterwards? Dr. Reece's report gives a clear answer. Before January, 1903, the central part of the city, and also—and especially—its north-eastern part near to Priory Road Hospital, had been principally affected. Cases had occurred from time to time in the area within a mile of the (as regards small-pox) empty Park Hill Hospital; but relatively to the rest of the city, the incidence of small-pox on this area had been slight. Park Hill Hospital was opened on January 12th, to meet a sudden demand for extra isolation, entailed by a localised outbreak in the central part of the city, due to an unnotified and severe case which was moribund when discovered. The admissions to Park Hill Hospital in successive fortnights ended January 17th and 31st, February 14th and 28th, were respectively 7, 88, 55, and 97. The houses newly invaded in the inhabited area within a mile of the hospital, in the fortnights ended January 3rd, 17th, 31st, and February 14th, were respectively 4, 3, 2, and 3. Then, in the fortnight ended February 28th, the number of invaded dwellings in the mile area rose to 31; and in the three following fortnights 23, 55, and 40 houses in this area were newly invaded by the disease. And until Park Hill Hospital ceased to receive cases of small-pox, in June, this area continued to suffer to a far greater extent, relatively, than the rest of Liverpool. This is shown graphically in Dr. Reece's diagram, exhibited.

In the matter of graduation of incidence of small-pox, again, the familiar experience was repeated. The following are the figures; the rates of invasion at different distances from the hospital are also shown on Diagram II, extracted from Dr. Reece's Report.



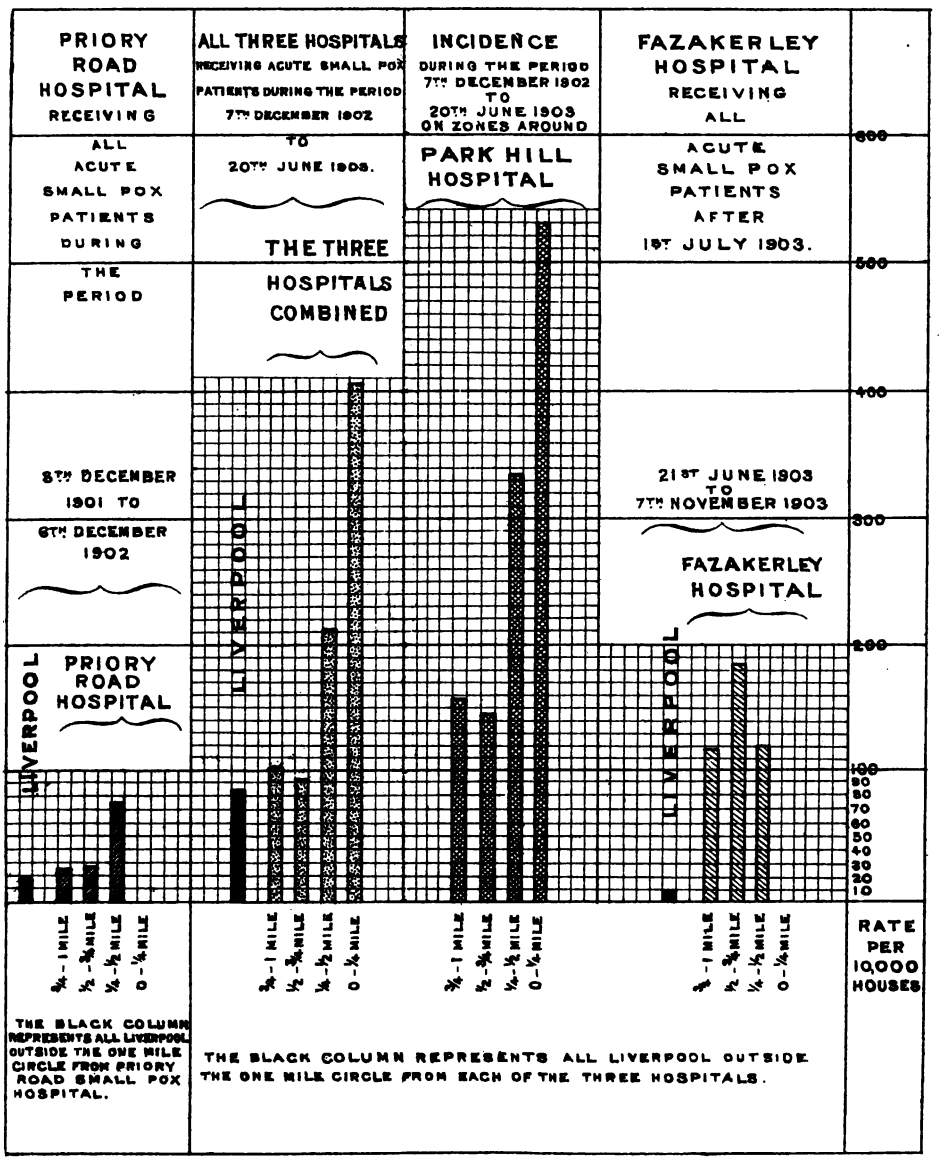
FIFTY CONSECUTIVE FORTNIGHTS COMMENCING 8TH DECEMBER 1901.

0046 R.O.S.

Weller & Graham, Ltd Litho. London.

DIAGRAM II.

SHOWING IN SUCCESSIVE PERIODS, DECEMBER 1901 TO NOVEMBER 1903, THE INCIDENCE OF SMALL POX INVASION, PER 10,000 HOUSES IN EACH INSTANCE, IN THE SEVERAL AREAS WITHIN ONE MILE OF LIVERPOOL SMALL POX HOSPITALS, AND ON CERTAIN SUB-DIVISIONS OF THOSE AREAS. FOR PURPOSES OF COMPARISON THE "INVASION RATE" IN EACH PERIOD OF THE HOUSES OF LIVERPOOL AS A WHOLE IS SHOWN IN EACH INSTANCE BY A BLACK COLUMN.



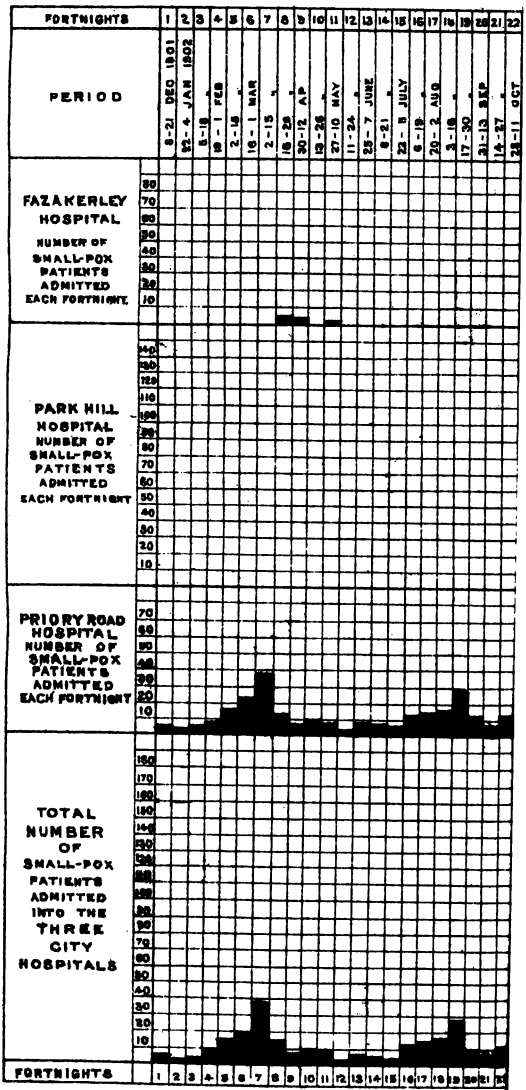
6062. 12.05.

Weller & Graham: Ltd Litho. London

DIAGRA

SHOWING FORTNIGHT BY FORTNIGHT FR

1903 THE NUMBER OF SMALL POX PATIENTS RECEIVED



604612.05.

December 7th, 1902, to June 20th, 1903.

—	Distant from Park Hill Hospital.				Liverpool more than one Mile from any Small-Pox Hospital.
	0 – ¼ Mile.	¼ – ½ Mile.	½ – ¾ Mile.	¾ – 1 Mile.	
Total houses ...	171	2259	4563	5617	100,969
Number of houses newly invaded by small-pox ...	9	75	67	89	868*
Houses newly invaded, per 1,000 of total houses	53	33	15	16	8.6

As regards the other two hospitals, it will be seen from Dr. Reece's tables and diagrams that, during the first year, 1902, when the smaller Priory Road Hospital sufficed for Liverpool cases, the incidence upon dwellings within a mile of that hospital exceeded that on the rest of Liverpool, including that on the mile area round the two hospitals, Fazakerley and Park Hill, which then were not receiving any small-pox cases. From December 8th, 1901, to December 6th, 1902, the invasion rate was only 1.6 per thousand houses in the city more than one mile away from Priory Road Hospital; while it was 2.76 on houses situated within a mile of the hospital, 3.33 on houses within three-quarters of a mile of the hospital, and 6.81 on houses within half a mile of the hospital.

I may refer to two other points which come out from study of the fortnightly maps given in this Report. The first is, that in the early part of the second year, 1903, after both Park Hill and Priory Hospitals had become full of small-pox cases, the bulk of the city, which is more than one mile from either hospital, is seen to have been affected to a much less extent than either of the areas within a mile of these hospitals. The second is, that towards the end of the epidemic, in the second half of 1903, small-pox, which was dying out in the city, and dying out of the Park Hill and Priory Road Hospital areas, lingered to a very noticeable degree in the comparatively sparsely-populated areas

* Two hundred and ninety-six of these houses were invaded between 7th December and 14th February, during which time only twelve houses in the mile area were invaded. The hospital did not receive any small-pox cases before 12th January, 1903.

round Fazakerly Hospital, which during that time had been receiving the whole of Liverpool's small-pox cases.

The history of these two epidemic years in Liverpool, as given in Dr. Reece's Report, affords a striking and recent illustration of the way in which, in epidemic years, the local distribution of small-pox in a large inhabited area may be determined by the operations of small-pox hospitals situated within or on the outskirts of that area.

General Review of Small-pox Hospital Influence, 1900-4, in the Above Cases.

Let us consider the facts above summarised regarding the epidemic period 1900-4. In one instance, Manchester, which must be considered with the reservations above noted, the evidence of hospital influence is negative. But in the other four cases which I have reviewed, definite positive evidence of hospital influence has been forthcoming. Clearly, the Hospital Ships in Long Reach, the Hospitals of Belvedere, Sheriff Hill, Park Hill, and Priory Road, have exerted a very conspicuous influence in enhancing the prevalence of small-pox respectively in the Orsett Union, in Glasgow, in Gateshead and Felling, and in Liverpool.

Besides Manchester, there may have been other towns in the kingdom during 1900-4 in which there has been a notable prevalence of small-pox, in which small-pox cases have been received in a hospital or hospitals so situated in regard to populous areas as to permit the matter to be tested, in which it has actually been tested by careful study, in which the facts have been exactly recorded, and have proved negative as regards hospital influence. If such cases exist, I regret that I have been unable to find them, and, consequently, have not included them in my paper.

Obviously, the Manchester case, together with other recorded instances (if such exist) that are negative in the above sense, demand careful consideration and comparison with the numerous positive instances. But they do not in least take away from the importance of these positive instances. No one would claim that contamination by typhoid excrement of a stream on a gathering-ground must always be followed by an epidemic of typhoid among the water consumers. Conversely, no one would claim the escape of the consumers from such an epidemic.

as evidence that water contaminated by typhoid excrement cannot cause typhoid outbreaks. Whatever explanation we give of it, we are bound to accept the fact that when hospitals used for acute cases of small-pox have been situated in or near to populous areas, they have frequently had the effect of starting, augmenting, or maintaining the prevalence of small-pox in those areas. The areas in question have been influenced in a special fashion, characterised by an incidence of small-pox on their dwellings, which diminishes in intensity with the distance from the hospital, and has been traced as far as a mile away. The influence exerted in the above circumstances has again and again been one of the principal facts, if not the dominating fact, of a small-pox epidemic, and thus has entailed very serious consequences to the public health.

We cannot get away from these facts; they are as definite as any known to epidemiology. When Dr. McVail read the paper to which I have alluded, in 1894, they had already been ascertained by a multiplicity of careful and detailed observations, in respect of many hospitals, in different epidemics, in London and the provinces. Absence of small-pox, and the general removal of small-pox hospitals to sparsely-populated areas, led, perhaps, to this having been to some extent forgotten a few years ago. Recent epidemics have now enabled the question to be tested afresh, with results entirely confirming those of former years. "Hospital influence," and its explanation, must remain one of the most important chapters in the natural history of small-pox.

Explanation of "Hospital Influence."

To come now to the explanation of the facts, and their bearing on the thesis of aerial convection. Any one who studies Mr. Power's writings of twenty years ago on this subject, will find that the principal basis of his thesis of aerial convection consists, not—as is sometimes wrongly stated—of an elimination of every other possible cause of spread of small-pox in the neighbourhood of the hospital, but in the explanation which it afforded of one of the principal manifestations of this hospital influence—the characteristic graduation in the incidence of small-pox on the neighbourhood of the hospital. Now, in 1900-4 we have again had striking evidence of the existence of this graduation occurring in different epidemics round a plurality of hospitals. Again it has to be accounted for, and the

conditions of inquiry demand that the explanation should be consistent, and one which is applicable to each of these hospitals in each of these epidemics.

Graduated intensity of small-pox incidence has never been explained by traffic of vehicles and ambulances to and from the hospital, by communications of patients in the hospital with other persons outside, or by incomings and outgoings of staff. Nor do such explanations assist in regard to the hospitals of 1900-4. In none of these instances is there any evidence that the staff was recruited from the immediate locality of the hospital: we all know that a hospital staff is not obtained in this way. Nor is there any evidence that the visits of the staff to shops or to houses of friends were graduated in frequency according to the distance of these houses and shops from the hospital. In Gateshead I obtained direct evidence in the contrary sense: when the staff left the hospital (which happened comparatively rarely), their custom was to go straight down to the busy centres and shops of the town, or over to Newcastle. Similarly, it is not the custom of municipalities to select the contractors who supply the hospital with food or stores, according to the proximity of their houses to the hospital. So, too, with regard to visitors or friends of patients, coming to the hospital, looking over the wall, through the gates or railings, and the like. In the 1900-4 cases, where such authorised or unauthorised visits were made, I can find no evidence that those who came most frequently were persons dwelling within a quarter of a mile; next, those who dwelt between a quarter and half-mile from the hospital, and so on. Consider, for example, the case of Belvedere Hospital in the two periods of recrudescence. Here, on each occasion, the small-pox cases first admitted to the hospital came from a part of Glasgow some miles from the hospital; and it is impossible to account for the outbreak which in each case followed in the hospital neighbourhood on any assumption of visits of friends or relatives. The suggestion has been made that mere curiosity of the inhabitants living near to a small-pox hospital would encourage them to try to communicate with patients. Possibly, for a time, or on some special occasion; but on what grounds can we assume such persistent and graduated curiosity continuing month after month and year after year? Gross maladministration, a staff incompetent or improperly supervised, patients getting out of hospital grounds inadequately enclosed, might entail local spread of small-pox due to personal communication among persons

living nearest to the hospital. Such opportunities were afforded the other day in the case of Dewsbury Hospital. But who can suppose such gross maladministration at Glasgow, or at Liverpool? In such cities we have not gone back in our hospital organisation or in our precautions during the last twenty years; we have advanced.

Ambulance traffic has specially to be considered. It has been suggested that the small-pox patient in an ambulance gives off particulate infection of small-pox into the air, and that aerial convection for short distances of these infective particles, operating on a susceptible individual during a very short period of time, would be more likely to give him small-pox than would infective particles derived from an aggregation of acute small-pox cases, and conveyed through the atmosphere from a greater distance. On this assumption, the concentration of ambulance traffic towards a given hospital might be expected to occasion an exceptional invasion of dwellings near to the hospital, and also might, in some measure, explain graduation of incidence. But, as Mr. Power showed, we should expect, if this were so, to find the invaded houses grouped along, or near to, main lines of communication and traffic to the hospital, and this he found not to be the case. I cannot do better, also, than quote Dr. McVail's observations on this point: I am unable to find any evidence from the experiences of 1900-1904 which points to a different conclusion:—

"It is true that people living in one street might encounter the ambulance in another, and go home and lie down with the disease a fortnight later. But it is equally true, that on any sufficient basis of facts, a larger proportion of people belonging to the streets traversed by the vans would be exposed to the ambulance influence, than of people belonging to other streets. And, in regard to those who met the van in one street and lived elsewhere, it would be a very extraordinary thing if the houses to which these people returned happened to be arranged in numbers diminishing regularly according to the distance from the hospital to which the van was travelling. It would be still more extraordinary if this same regularity of arrangement were found to repeat itself around the same hospital in epidemic after epidemic."

We must admit, then, as regards the 1900-1904 experience, that we cannot fix responsibility on to any single component of "hospital communication and traffic," any more than we have been able to do so in the case of Fulham in 1885, or in other subsequent instances. We cannot say it is the ambulances, or the staff, or the patients' friends, or the tradesmen. But it is worth considering whether the sum of all these possibilities of infection can account for the influence of the hospital on its neighbourhood—direct personal communication with a patient here, indirect infection by a nurse there; an ambulance on this occasion; a

wall-climbing patient on that, a porter's visit without disinfection on a third. True, the graduation of incidence is not explained in this way, but may it be that in some manner or other not yet understood the total effect of all these things may give us graduation?

My own observations as regards two of the hospitals which spread small-pox in 1900-4 point to a negative answer to this question. The hospitals are Sheriff Hill and the Hospital Ships. Sheriff Hill Hospital is close to the boundary of Gateshead and Felling. During the epidemic of 1900-4 all the small-pox cases in this hospital came from Gateshead. Felling had its own hospital more than two miles away. No ambulance came to the hospital from or through Felling; the friends of patients and others who came to the hospital, came, not from Felling but from Gateshead. The staff of the hospital had nothing to do with Felling, and never went there. The Felling people at Windy Nook, who suffered severely and persistently during the epidemic, had no curiosity as regards the Gateshead patients in the hospital on the hill above them; it was true that many of their friends had small-pox, but these were at Felling Hospital. Windy Nook people, going into Felling, or to Gateshead, or to Newcastle, would have to go out of their way and up-hill in order to pass the Sheriff Hill Hospital. In short, the sum of "hospital communication and traffic," in regard to Felling, was practically nil. Nevertheless, as my report shows, the exceptional incidence of small-pox was more conspicuous on the Felling side of Sheriff Hill Hospital than that on the dwellings in Gateshead, at corresponding distances from the hospital.

The Hospital Ships in Long Reach during 1901-2 had abundant communication on the Kent shore with Dartford. They are moored off the Kent shore, and all their land traffic and communications are with this side. Neither ambulance steamers, patients, nor staff had anything to do with Purfleet, half a mile away across the water. Hospital communication and traffic in this case was non-existent. Anyone acquainted with Purfleet, and who has also lived on the ships, as I have done, knows that there is not the least attraction in that place, and that the staff has nothing to gain by rowing across there, even if the ships' rules allowed it. I have been told that the statement has been made that in 1895 members of the staff did sometimes row across to Purfleet, and that it has hence been inferred that in 1901-2 there may again have been such communication. I claim, however, in this case to speak with the authority

of most careful local inquiry on many occasions throughout the latter epidemic. Moreover, if any such visits occurred, they could afford satisfactory explanation of the Purfleet epidemic only on the assumption that they were made systematically week by week, or month by month, throughout that epidemic; that members of the staff were able to get boats for this purpose, and so systematically to transgress the hospital rules; that on each occasion they further transgressed the hospital rules by neglecting to change their clothing and carry out the prescribed rules for disinfection; and that on each occasion they landed at Purfleet, left their boat and mixed with its inhabitants without it being known who they were, or whence they had come. Such assumptions have no basis in fact.

Nor does the negative experience of Manchester help us in this connection. The factors which I have enumerated existed in Manchester as elsewhere. Ambulances converged to Clayton Vale Hospital from all parts of the city; the staff was not kept for two years within the walls of the hospital; Dr. Niven specially notes that people came to the hospital on Sundays, looked into the grounds, and all the rest of it. No doubt, as a whole, the administrative precautions taken by Manchester against spread of infection by hospital communications and traffic were excellent—this may be expected of Manchester. But what is the evidence that they differed, to any material degree, from those of Liverpool or Glasgow? It is hard to say which of these three great cities we should select if we required to show instances of excellent modern administration, in the matter of ambulance service, hospital precautions, and the like.

We have now to consider the recognised explanation of "hospital influence"—the hypothesis of aerial convection—in relation to the behaviour of small-pox during the 1900-4 period in dwellings in the neighbourhood of the several hospitals under consideration.

Considerations as to "Aerial Convection."

Aerial convection, as an explanation of "hospital influence," requires it to be granted, in the first instance, that the infective material by which small-pox infection is transmitted is particulate; that the air of hospital wards in which acute cases of small-pox are isolated contains, often or always, an abundance of infectious particulate matter; and that these particles may be carried up into

the outer atmosphere by currents of warm air rising from the hospital.

In these circumstances, the fate of the floating particles will be determined by the prevalent conditions of the atmosphere: wind, temperature, moisture, and the like. It may be presumed that when (as will most usually happen) they meet with a definite wind of moderate or strong velocity, they will thereby, in ordinary cases, become spread out and scattered, so that the individual particles, by the time they have ultimately fallen, are, so to speak, anywhere; the greater part of them, most likely, having been carried far away from the neighbourhood of the hospital. Again, if the escaping particles are carried from the hospital into a shower of rain, they will presumably be cleared out of the air and washed to the earth. The theory of aerial convection, as applied to explain the characteristic incidence of small-pox round the hospital, requires that from time to time meteorological conditions will enable the floating particles to be carried by light air currents for variable distances, in such a way that the floating matter undergoes no great degree of dispersion, and tends to settle over comparatively limited areas in the neighbourhood of the hospital. The situation of these areas in regard to the hospital will in such circumstances be governed by the character and direction of the prevailing convection currents. The result is that, now and again, and here and there, persons living in the vicinity of the hospital become exposed, it may be for a few minutes or for a few hours, to the inhalation of air which, in regard of the floating particles it contains, approximates in infective ability to that escaping from the acute small-pox ward of the hospital.

It will, I think, be readily admitted that a hypothesis of aerial convection operating in some such manner as this affords (what we cannot otherwise obtain) a consistent explanation of the facts of 1900-4 with regard to all the hospitals which spread small-pox in their neighbourhood. If accepted, it accounts for Purfleet and Felling as well as for Liverpool and Glasgow. It explains the transference of the disease from one quarter of the city to another, as a result of the hospital operations of Glasgow and Liverpool. It explains the persistence and recurrence of "hospital influence" during period after period of small-pox prevalence. It enables us to understand how it is that this hospital influence has continued to be exerted in large cities, notwithstanding modern sanitary organisation and

modern improvements in hospital administration. Especially, also, it enables us to comprehend the occurrence of graduation in intensity of small-pox incidence. And, finally, this hypothesis is the only consistent explanation of the instances of "hospital influence" in former years; and thus we have a means of reconciling present and past experience in this respect.

It should be added, also, that the hypothesis of aerial convection gives us a suggestion of the reason for the escape of Manchester. Dr. Niven (who some years ago had experience of spread of small-pox round the Westhulme Hospital of Oldham, and also, I understand, at the Monsall Hospital of Manchester) adopted at Clayton Vale Hospital, Manchester, both in 1902-3 and in 1903-4, methods specially designed to reduce the access of particulate infectious matter to the air of the hospital. Great care was taken in the cleansing of the wards, so that the amount of dust was reduced to a minimum. Every case of small-pox in the acute stage was oiled, twice daily, with a 2.5 per cent. solution of carbolic oil. In special and severe cases, extensive portions of the body, or even the whole body. were kept swathed in lint wrung out of boric solution and enclosed by gutta-percha tissue, while a mask of the same kind was placed over the face—other parts of the body not so dressed were kept oiled. It should be noted that this system, the persistent performance of which is, in Dr. Niven's words, an unpleasant and tedious work, requiring both energy and determination, was much more than the mere limited application of carbolic oil or dressings to certain cases, which is practised in many small-pox hospitals. As I have said, the smallness of the population living within half a mile of Clayton Vale Hospital, and the comparatively limited proportions of the epidemics in Manchester, makes caution necessary in drawing conclusions from the absence of "hospital influence" there. But it is at least very suggestive that the negative results of Manchester are met with in the case of a hospital where special care was taken to prevent aerial convection of particulate matter.

During the many years in which the thesis of aerial convection has been available, and has been discussed as an explanation of "hospital influence," it has not been objected to, so far as I am aware, on the ground that it involves unlikely or extravagant assumptions. At the same time, I have occasionally found colleagues in the public health service who are not only sceptical as to the

existence of aerial convection, but who also go so far as to prefer leaving hospital influence unexplained to accepting aerial convection as the explanation. For example, they will refuse to consider the possibility that a given case of small-pox in the neighbourhood of a small-pox hospital has been caused by aerial convection so long as there is any chance, however remote, that the patient contracted his small-pox (apart from any relations with the hospital) by "direct" or by "mediate" infection, or by exposure to a hypothetical "missed case." On these lines, particularly if the term "mediate" is made sufficiently elastic, they can without much difficulty dispose of nearly every case in the hospital neighbourhood. But, having done so, they leave us in a worse position than before: a position in which it is necessary to assume that the causes of direct or "mediate" infection—the tramp, the contact of the contact, the "missed case," and the rest, are more dangerous to people who happen to live in the neighbourhood of the hospital than to those who live elsewhere.

This untenable position no doubt in part is merely the result of their having been unable to see the wood for the trees. But the basis of it is, probably, an impression that the suppositions involved in aerial convection are, *a priori*, improbable. It may be useful, therefore, briefly to inquire: Do the assumptions of the aerial hypothesis conflict with present-day knowledge as to the nature of small-pox infection, or as to the behaviour of floating particles in the air?

The infection of small-pox is certainly particulate. We do not know much regarding the micro-organism of variola, but there is no question that it must be present in abundance in the secretions and exudations of the mouth and air-passages, and in the vesicles on the skin, of an acute case of small-pox. Dr. Mervyn Gordon's recent work on salivary bacteria helps us to realise the enormous number of minute particles of infectious saliva and mucus which must be projected into the air when a patient who has abundant eruption, accompanied by copious secretion, in his mouth and throat, shouts, sneezes, or coughs. It is inevitable, also, that minute particles of epidermis, fresh from contact with virulent small-pox lymph, must be shed in immense quantity into the atmosphere of a small-pox ward, particularly when patients are restless or struggling. There is an obvious difference between acute cases of small-pox and acute cases of scarlet fever in the degree of their ability to cast infective particles into the air. If asked as

to the relative danger incurred in exposing susceptible persons to the atmosphere of a ward containing acute small-pox and one containing acute scarlet fever, general experience of both diseases would justify us in attributing the greater danger to small-pox. It should be added also that there is ample evidence that the infectivity of the small-pox virus is not destroyed by dessication.

An abundance of floating infectious particles in air escaping from a small-pox ward may then reasonably be assumed. It is needless to dwell on the ability of fine particulate matter to float in the air and to be carried by air-currents, or upon its tendency to fall. We need not go for illustration to Tyndall's experiments, or to Krakatoa, or to Mont Pelée: observation of the household chimney will suffice. If biological illustrations are desired, there is the experiment of Hutchison, who sprayed *B. prodigiosus* into the open air of the parade-ground at Göttingen, and recovered the bacilli on plates 170 and 600 metres away. Dr. Gordon informs me that he has found evidence of the convection of microscopic droplets of saliva to at least 150 feet from the mouth of the speaker in a large assembly-room.

The important question is, of course, the ability of these floating particles, in suitable conditions, to settle in the neighbourhood of the hospital without a great degree of dispersion. Here, again, we can form some judgment of the probability of this occurrence from familiar observations. Instances are known in which weighable quantities of metallic deposit, consisting of fine solid particles derived from zinc- or copper-smelting works, have been obtained from leaves and foliage collected many miles away from the chimney responsible. Again, in the case of alkali works, minute floating particles of moisture, saturated with acid gases, are carried long distances in the air before they fall; and it frequently happens (particularly on cold nights, accompanied by very gentle breezes) that the air-current containing these acid particles pitches, as it were, on a limited area a mile or two away from the works, producing a strictly localised destruction of vegetation. In such cases, the alkali company does not question aerial convection: it pays compensation. A more familiar instance, and perhaps a better analogy, is a factory chimney giving out smoke on a comparatively calm day. The eye can trace the smoke particles, along lines of steady air-currents, in their ascent, in their deflection from the chimney, and in their descent. The smoke can be watched as it nears the ground; it may

be close to the chimney or a mile away from it, spreading out and depositing its particles over a comparatively small area. It may be remembered, in connection with this analogy of a chimney, that the volume of air escaping from the wards of a small-pox hospital is usually large, and that the difference in temperature between the hospital and the outer air is often considerable. The effect of this temperature difference, on a calm day, would be to carry floating particles in the first instance to a considerable height above the hospital.

The chimney analogy is also useful in considering the limitations which the facts require us to place on the operation of aerial infectivity. Individual dwellings, groups of dwellings, or institutions, may be in the neighbourhood of the factory chimney, even close up to it, and yet the accidents of air-currents have never, throughout several months, brought them at any one time more than a few unnoticed soot particles. In other dwellings, or groups of dwellings, it may happen perhaps twice in one day that the inhabitants have cause to ask "where these blacks are coming from?"

Much more could be said on this subject, but the above considerations, I think, sufficiently indicate that the assumption that, on occasion, particulate matter from the hospital descends in the neighbourhood without a great degree of dispersion is in no way exacting. There would, indeed, be strong grounds for disputing a contrary proposition.

From *a priori* considerations, however, we should expect these occasions to be relatively infrequent. There are many obvious reasons which make it impossible to believe that they continually arise, hour by hour and day by day, in all states of wind, temperature and moisture. This point, though self-evident, is important: the theory of aerial convection, as an explanation of the known facts of small-pox hospital influence, requires that the occasions on which infection can be carried aerially should be relatively infrequent. This consideration, though fully dealt with by Mr. Power, appears to have been missed by some writers on the subject, who seem to regard acceptance of aerial convection as implying that at all times, when a hospital is receiving acute cases of small-pox, infection is being conveyed aerially to all parts of the hospital neighbourhood. For these writers, acceptance of aerial convection would apparently require us to believe, contrary to experience, that a susceptible person merely passing a small-pox

hospital, in a vehicle or on foot, thereby incurs something worth calling a "risk" of contracting small-pox. In the same way, they appear to claim that aerial convection, if it occurs at all, must, during an epidemic, affect every house and every institution near the hospital. They are not content with a house-invasion rate of 5 per cent.; they demand that it should be 100 per cent. Having made that demand, they usually point to the escape of the 95 per cent. as evidence that aerial convection has not been operative. And, in conclusion, they say that Mr. Power's theory "has been greatly exaggerated." The exaggeration is all their own.

If, on the theory of aerial convection, the infectious ability of a hospital in regard to its neighbourhood occurs only from time to time, and in suitable meteorological conditions, we have still to ask, in order to complete our inquiry, whether such suitable conditions did obtain on occasions when aerial convection was operative round the hospitals of the period 1900-4. Unfortunately, this question cannot be answered with any certainty. The difficulties in ascertaining, on the one hand, the facts regarding the date and hour of infection of a given case; and, on the other hand, the meteorological conditions of that date and hour, are insuperable. All that can be done is, where possible, to fix upon certain days on which aerial convection appears to have been specially manifested, and then to ascertain the general weather conditions of those days, as recorded at the nearest observatory. Some facts are recorded which enable this to be done in the case of Belvedere Hospital in 1900 and 1901, and in the case of the Hospital Ships at Long Reach in 1901-2. Examination in the above sense of the limited data available indicates, in both instances, that when aerial convection appears to have been infecting persons living near the hospital, the general type of weather was consistent with periods of calm or light winds, sometimes accompanied by fog. I have not met with any instance in which the days coming specially under suspicion were characterised, according to the records available, by persistent strong winds or by practically continuous rainfall.

The question can also be approached in another way, by inquiring whether the direction of prevalent light winds accords with the position, as regards the hospital, of neighbourhoods which have been specially affected. In this connection I would note that in Long Reach light winds blowing from west-south-west to east-north-east, *i.e.*, from the Hospital Ships towards Purfleet, are apparently fre-

quent. In the case of Sheriff Hill Hospital, also, it was noteworthy that Windy Nook and other parts of Felling which were specially affected by small-pox, lie in the path of south and south-west winds, blowing from the hospital; and that winds from this quarter are here frequent and are commonly light.

In brief, the conclusion of this paper is, that the experience of the recent epidemic period, 1900-4, has again afforded us a remarkable demonstration of "small-pox hospital influence"—a demonstration wholly in accord with the experience of previous epidemics. For the consistent explanation of this "influence," we cannot, in our present knowledge, do without the thesis of aerial convection. On the contrary, the case for accepting this hypothesis has been strengthened. We have reached a position, indeed, in which we cannot be content with any mere negation of aerial convection, and cannot pass the matter by with the convenient phase, "Not proven." Epidemiology, as a science, requires us to continue to accept it, and to act upon it, as a satisfactory working hypothesis, until we can be shown some other and more probable explanation, which is applicable with equal consistency to the numerous and varied occasions on which hospital influence has been manifested during the past five-and-twenty years.

Discussion on Dr. Buchanan's Paper.

Dr. C. B. Ker: I should like, in the first place, to say with how much admiration I have listened to Dr. Buchanan's Paper. I am bound to admit that I have listened to him to-night feeling much more predisposed in favour of aerial convection than hitherto. I own frankly that my views have always before been rather contrary to this hypothesis. But I do not wish to make too many criticisms: I thought it would be interesting to give you just the personal experience we have had in Edinburgh during the small outbreak we had last year.

The Edinburgh Hospital is, I regret to say—and I say it with all due deference—deliberately situated in defiance of every recommendation of the Local Government Board. It marches with the ordinary Fever Hospital for Edinburgh—that is to say, the grounds of the two hospitals adjoin. This hospital has a population, including staff and patients,

of about 450 people. On the other side is one of the poorhouses for the city of Edinburgh, which has a population of about 1000 people; while to the back of the hospital is a hydropathic establishment, which is a place of large resort, and has a population of about 200 people. Now, all these places have their boundaries practically adjoining. The distance from pavilion to pavilion of the Small-pox Hospital and the Fever Hospital is a matter of 72 yards; the nearest point of the actual buildings of the poorhouse to the nearest pavilion of the Small-pox Hospital is about 325 yards. The paupers in the poorhouse, many of whom are employed in agricultural work, work right up to the boundary of the Small-pox Hospital, which is quite close to the Small-pox pavilions. During the time of the outbreak a large number ef these paupers were engaged close up to the hospital boundary, and a large number of general patients in the Fever Hospital were equally close on the other side.

In addition to this, if you take a larger area, you come to some other institutions. There is the Craig House Asylum, with 325 people. Within the mile area, and in the direction to which the prevailing wind blows, there is a population of 3000 people. In the other direction there are two villages, one with a population of 520 people, and the other with a population of 730.

The interesting point is that, with the exception of the poorhouse, there were no cases in this area. Dr. Harvey Littlejohn investigated three cases in the poorhouse, and he assures me that these were quite satisfactorily explained, two of them coming from infected lodging-houses. The other poorhouse, moreover, four miles away, had the same number of cases. With the exception of those cases, there was only one case within a mile of the hospital. The hospital was open for some time, but it had not a very large population. The average number of small-pox patients for a period of two months was about fifty-five. But Dr. Buchanan has pointed out that, with an average no larger than that, the influence was felt around Belvedere. Again, I gather that there were only sixty patients in Priory Road Hospital, in Liverpool, when the influence began to be felt. Therefore, I assume that we must allow that fifty patients can be dangerous; and if that is the case, it is an interesting fact that we had this population of fifty-five for two months consecutively (and if you take the three months, the average was forty-seven patients), and yet there was no case of small-pox within a mile, even with this considerable population.

A MEMBER: What about vaccination?

Of course, the Fever Hospital staff is adequately vaccinated, and most of the patients, being children, recently vaccinated. But the pavilion that stands within 72 yards of the small-pox pavilion is the erysipelas pavilion, which contains nothing but adults; there are no children in it. It also contains a class of patients of the lowest type, and the least likely to be re-vaccinated. I don't think anyone would say that you could safely vaccinate an erysipelas patient. Not a single precaution was taken during these three months, and the erysipelas wards were full all the time.

The next nearest were the typhoid wards; we took no special precautions, except as regards the staff. In the same way, there were no special precautions taken in the poorhouse, except in the case of the persons who had been in contact with the cases that occurred.

The population towards Edinburgh in the direction of the prevailing winds is about 3000 people, and embraces a pretty good residential district. Of course, we pride ourselves on being better vaccinated than most large English towns, but that is a sufficient population to contain many people not sufficiently re-vaccinated. I say that is an interesting negative case. The question naturally arises: Is there any reason at all why this should be? Is there any difference in the way, for instance, in which the Edinburgh Hospital is managed, or anything to account for this in any way? The only thing that I do differently from other people is this: we have one rule with regard to the nursing staff which we keep absolutely rigidly. The nurses in the Small-pox Hospital are not allowed to go beyond the hospital grounds, in any circumstances whatever. Every nurse in the hospital volunteers for small-pox service, and she remains for a period of six weeks. She knows when she goes to the Small-pox Hospital that she will not be allowed to leave it until the six weeks are up, unless she breaks down. Only the best nurses are allowed to go—the most capable and trustworthy—and it is considered an honour to be allowed to go. No one goes in and out of that hospital except the medical officer. I am the only person who enters and leaves the hospital, except the ambulance attendants. This probably does not amount to very much, but that is the only thing in which we differ from the majority of other small-pox hospitals. That is the advantage of having your small-pox hospital near to your ordinary fever hospital; you can select your nurses in

this way. We do not get together a haphazard staff for an epidemic, as is often done; it is a privilege which only the best nurses are allowed to partake of. Of course, there is always a chance of leakage. I do not think in other ways we manage better than other people; but we put nurses whom we know are, at any rate, particularly conscientious and suitable people to put there. In that way, we have perhaps a better chance of preventing leakage. Of course, one has seen dreadful things in different hospitals. In the old hospital it was, on one occasion, necessary to have a cordon of police to prevent people shaking hands over the walls. In our present hospital they cannot possibly do that. We have more control than we used to have.

Just outside the mile limit there were about three or four persons infected, and in three out of the four cases there was what I might call satisfactory exposure; in the fourth case we could not trace it in the least. I do not know how it came, but in any case that house was just over a mile away; it was a very difficult case to explain. If the infection was aerial, it had passed harmlessly over the heads of 4000 persons to reach the infected house.

The only other point that I will trouble you with—or, perhaps, I should say the only deduction I should like to draw—is: Are not the regulations of the Local Government Board a little vexatious? I think they actually mention poorhouses and fever hospitals, but here we are actually wedged in in a strategical position between these three public institutions; and yet, as far as we can make out, with an average of fifty patients in our hospital, there has been no spread in its neighbourhood. I am prepared to admit that if we ran up to a couple of hundred, as at the hospital ships, it might be different. But why should we prepare for a large epidemic? If you isolate rapidly, and vaccinate fully, surely fifty is enough to start. Why should unfortunate municipalities have small-pox hospitals at the end of the world, where they cannot be properly supervised? You cannot get supervison with a temporary staff, and temporary doctors, and everything else temporary. You can for 150 patients, because you have then to get a big staff; but I am thinking of the chance of leakage in a small hospital of ten, or fifteen, or twenty cases, put right out into the country. It seems to me that it is much better to have them under the eye of some competent person nearer to the town.

I have brought a map with me, because I thought it might interest some members of the Society, as showing

the locality in which the hospital is situated. It is an ordnance map of the city of Edinburgh, on which the old and the new hospital are both shown. I was formerly in charge of these buildings of the old hospital, which was in the centre of the city of Edinburgh.

We used to be abused for causing small-pox in this neighbourhood where our old hospital stood; but note that the cases are in the same neighbourhood now, and that they decrease in numbers the further we go from this hospital, empty though it was. There were no special antiseptic precautions taken. I do not think we took any special measures in that way. The dust is destroyed, but, of course, a great deal must escape.

In conclusion, I do not in any way wish to deny the possibility of air-borne infection. My contention is that, for practical purposes, it may be neglected. It is not the best way to secure efficient administration of small-pox hospitals to allow those in charge of them to plead aerial convection, if the hospital influence is at all felt. The acceptance of the theory is bound to weaken the sense of responsibility in even the most conscientious of us.

DR. NEWSHOLME: I am sure that, in the first place, we shall agree in heartily expressing our appreciation of Dr. Buchanan's Paper: which shows that he has, to a very considerable extent, inherited the happiness of phrase and lucidity of expression which always distinguished the writings of the late Sir George Buchanan.

My own position in the matter of distal aerial convection of small-pox is that of an agnostic. I have no prejudice against it; I do not, however, regard it as proved. I wait for confirmatory evidence, and I incline to think that such evidence of a satisfactory character will not be forthcoming. I believe in the occasional operation of "hospital influence." I agree that there have been instances in which there has been excessive incidence of small-pox around small-pox hospitals; but I believe that when this has occurred, and is not due to the accidental sweep of the epidemic, it is most likely to have been caused, not by distal convection, but by the same cause as has produced epidemics of small-pox remote from such hospitals, viz., personal contact.

My beliefs are open to correction by evidence; but we are asked by Dr. Buchanan not merely to modify the strength of our beliefs, but to admit that

"We have reached a position, indeed, in which we cannot be content with any mere negation of aerial convection, and cannot pass the matter by with the convenient phrase 'not proven.'"

To such an admission a stricter examination of the evidence is necessary than would be required for determining a question of mere increase or decrease of probability; and the matter is further complicated by the association of Mr. Power's name with this hypothesis of aerial convection. Had Mr. Power time to attend these meetings, he would be made to realise the depth of respect in which his work, as well as he himself, is held. It is extremely difficult to disagree with a hypothesis which has received his endorsement, and, at least in its scientific form, is his own creation. That an infected house is a potential source of infection, that a hospital is a house predestined to infection, must be admitted as facts justifying all practicable means for separating the hospital from the town, even though we may recognise that a hospital should have means of controlling its infectivity which a private house cannot enjoy. The question would therefore be scarcely more than academic, were it not for the facts that precautions such as would be necessary on a hypothesis of distal aerial convection may cost a price which, if the hypothesis were incorrect, and hospital infectivity were controllable by improved administration, might be spent to better advantage to the public health, which constantly calls for more sacrifices than an ordinary community is able or willing to make.

I think we shall take a clearer view of the relative probabilities of distal and proximal convection, if we look at the history of the theory of aerial convection as an explanation of the spread of disease. I need not do more than refer to the miasmatic theories formerly held with regard to cholera and malaria. The theory of aerial convection was held even with regard to syphilis; and so late as 1881 a similar hypothesis was put forward by the late Dr. Airy for diphtheria; and he suggested that this disease could travel aerially three, six, twelve, eighteen, and even possibly thirty miles, and brought forward a number of instances in which coincidences were more numerous than doubtful cases. Dr. Airy's summing-up is very interesting. While admitting that his arguments and instances do not suffice to establish the theory of aerial spread of diphtheria, he adds:—

"I think they are sufficient, in conjunction with earlier observations, to give the theory a right to be disproved, and pending disproof, to be provisionally accepted."*

* *Trans.* Internat. Med. Congress, London, 1881; *Dis. of Children*, p. 69.

I place this transference of the burden of confirmation or disproof of an important induction to those who oppose his views, in conjunction with a similar remark by Dr. Buchanan.* He says:—

"I am struck by the absence of evidence that the problem has been carefully studied, in instances comparable to those I have mentioned, with the result that no graduated incidence has been observed."

While Dr. Airy's hypothesis has never been subjected to the process of disproof which he challenged, it may be taken as common ground that it has not received the acceptance of epidemiologists; and I think that both writers have underestimated the order of probability which should be established in favour of a hypothesis, before a burden of disproof can be held to rest on those who are unable to accept it. In the case of a distal aerial convection, of which the operation is conditioned as described by those who accept it for small-pox, there is a special objection to the demand for disproof. Taking the hypothesis as stated by Mr. Power and those who have followed him, it requires for its operation the simultaneous presence of many conditions, some of which are even now imperfectly specified and inadequately recorded; and as a matter of historical fact in the criticism of this hypothesis, any attempt to lay stress on epidemics of which the figures have not conformed to its requirements has been met by the reply that some essential condition must have been absent.

Distal aerial convection has, in point of fact, been a favourite preliminary hypothesis in the ætiological discussion of many diseases; but with extension of our knowledge it has been discarded successively in regard to ague, cholera, diphtheria, and influenza. The instance of influenza is the more striking, because the hypothesis of aerial convection had held the field for several centuries, until the investigations of Dr. Franklin Parsons disproved it. Distal aerial convection to-day remains as the occasional explanation of the spread of one disease only—small-pox.

It is common ground that personal infection is the most important and most frequent means of spreading small-pox. The balance of probability compels us to believe that in the instances in which personal infection cannot be traced, it does nevertheless operate; unless we find, after the most rigid investigation, that there is proof to the contrary. If circles and zones showed graduated incidence of small-pox

* *Journal* Roy. San. Instit., vol. xxvi, No. 4, May 1905, p. 205.

in the population around a small-pox hospital, and if the hospital could be shown to be strictly self-contained, without traffic in or out, the case for aerial convection would be demonstrated, assuming that the circum-hospital infection were shown not to have occurred from other external sources. Unless there are strong practical reasons to the contrary, we are bound in the absence of proof to accept the conclusion to which the greater weight of probability attaches. In a completely self-contained hospital circumstanced like the Small-pox hospital at Edinburgh, as described by Dr. Ker, we should under the circumstances named above have no hesitation in accepting aerial convection as the method of spread, if spread occurred. But it did not occur; and at other hospitals from which spread apparently has occurred we have the choice between three suppositions:—(1) Aerial infection; (2) personal infection by means of patients or the staff of the hospital and the traffic to and from it; and (3) personal infection from others, absolutely independent of the hospital, and in the normal course of an increasing epidemic. I propose to confine my further remarks to the additional evidence which Dr. Buchanan has adduced from the experience of recent years.

As to the second means of spread, we know that its operation can seldom be excluded without difficulty, even in the best-administered hospitals. Dr. Buchanan commits himself to the statement that no surreptitious visits were made at Purfleet from the hospital ships, and he claims to speak with the authority of most careful local inquiry. I do not think that this opinion of Dr. Buchanan's, formed as it was on the basis of an inquiry made some time after the event, can prevail against the considerable body of independent evidence, including that of Dr. Thresh's report, that such visits occurred; and it must be remembered that those who broke the regulations in this manner would not be likely to have given to an inquirer information which would convict themselves. In a large hospital it is very difficult to prevent such offences. But even if the isolation of the ships at Purfleet had been complete, it must be remembered that the epidemic was in vigorous progress at Erith and other places having traffic with Purfleet before it declared itself in the Orsett Union; and it not only would be exceedingly difficult to show that the Purfleet infection was not originally imported by personal infection from surrounding districts, but no serious attempt appears to have been made to do so. As an explanation of the

Purfleet (Orsett Union) outbreak, which consisted from first to last of only 52 cases, the amount of infected traffic, whether from the ships or from other districts, need only have been small. Probably a dozen primary cases would have sufficed; and it seems to me impossible to say that the presence of infection imported to that small extent is excluded by any of the facts published as to this epidemic. The effect of such importation would have been the more powerful if some of the earlier cases had been—as were the first case at Grays and others at Erith—"missed" cases. The Purfleet experience is, in fact, an example of the manner in which an explanation of the facts is possible without recourse to the hypothesis of aerial convection: and it seems to me to illustrate fairly the view that, in the absence of an exact history of each individual case of small-pox, such as can rarely be secured by an investigation late in the outbreak or after its close, the question is one of choice between two conjectural methods of convection: of which one is known to be potent in the diffusion of small-pox, and solely responsible for the diffusion of many other diseases; while the other has been displaced even as a working hypothesis from the ætiology of all diseases except small-pox, and is supported largely in respect of that disease by the allegation—in the case of Purfleet seen to be ill-founded—that no other explanation will cover all the facts.

In one respect the case of Purfleet lacks one element of the reasoning by which the hypothesis of distal convection is supported. It is alleged that hospitals which are surrounded by inhabited houses exhibit their infectivity by determining an excess of cases as compared with other parts of the infected district, and that this excess shows a characteristic graduation in the number of cases within distances varying from ¼ mile to 1 mile from the centre. Purfleet has no such complete inhabited environment; but in other recent cases, of which Liverpool is the most conspicuous, it is suggested that this characteristic graduation of an excess of cases is shown as clearly as can reasonably be expected in a phenomenon in the production of which there are admittedly other contributory factors. In some of the cases on which stress is laid, I do not think that, even after making allowance for the interference of other factors, the correspondence is close enough to justify the description of the graduation as characteristic. It should be added that, *even if such a graduation were closely made out, it would give no help in determining whether the*

infection was conveyed aerially or by personal traffic. A group of infective particles despatched from a centre would fall with similar distribution, whether they were shed by a man walking along streets and calling at houses, or by a man in a balloon, or by a current of air passing over the same path; and the density of infection on any area would in each case vary directly with the distance from the centre of infection, and inversely with the superficial area and with the number of houses. It would, therefore, be necessary to eliminate the effects of hospital traffic as a contributory cause before any excess-incidence in the circum-hospital area could show the operation of distal convection. This elimination is the more difficult because, although the precautions in some cases may leave it possible for distal convection to occur, they may, on the other hand, be carried far enough (as apparently in the case of Manchester) to oppose a notable hindrance to the diffusion of infection from the hospital by any means of convection. The question, therefore, to which an answer can be sought with most probability of success is whether, in fact, any such excess has been shown; and it must be remembered that if such an excess is shown, it still remains possible that it is due to hospital traffic, and capable of being controlled by improved administrative precautions. I do not propose to examine all the figures quoted by Dr. Buchanan, because the consideration of those of Liverpool, on which he perhaps lays most stress, will indicate the difficulty which I find in accepting such statistics as warranting the inference that hospitals determine an excess of small-pox in their neighbourhood. Dr. Reece's report on small-pox in Liverpool presents twenty-three spot maps, showing a density of infection round the hospitals which is undoubtedly high as compared with that of Liverpool as a whole. Such maps are obviously not an accurate measure of relative infectivity, because the density of infection in an element of a district depends on the number of houses as well as on the superficial area. Taking these spot maps, however, as being intended to give a rough-and-ready clue to the distribution of the disease, it is necessary, in the first instance, in order to determine whether there is an excess incidence of cases in the circum-hospital area, to ascertain whether similar densities can be observed in other areas of those dimensions. Both results would, for accuracy, need to be corrected for density of habitations; but the use of spot maps assumes that the absence of such corrections does not prevent the drawing

of valid and useful inferences from the density per unit of superficial area. I think, myself, that they may properly be used in this way for preliminary inferences, subject to a reservation to which I shall refer later; and I have in like manner made a study of Dr. Reece's maps, with the object of seeing whether the circum-hospital density, as shown on the maps, was, in fact, beyond what existed elsewhere. I found that the infection-densities in the mile radius in regard to each of the two large small-pox hospitals were notably less than those within circles drawn from centres over a mile distant from each hospital; and I append the figures which are thus disclosed, compared with the circum-hospital figures.

Miles	New Houses Invaded.				
	0 to ¼	¼ to ½	½ to ¾	¾ to 1	Total.
Priory Road Hospital.					
1. In circles and zones from the hospital as a centre	1	30	61	121	213
2. Ditto, from a point over a mile W.S.W. of the hospital ...	44	138	215	182	579
Park Hill Hospital.					
1. In circles and zones from the hospital as a centre	9	75	64	88	236
2. Ditto, from a point over a mile N.N.W. of the hospital ..	47	124	132	190	493

(Dr. Hope informs me that when circles are drawn around the empty Netherfield-road Hospital, circles and zones showing graduated incidence are obtained.)*

It appears, therefore, that in Liverpool the area on which, as shown by the test furnished by spot maps, the maximum density of infection—or, at least, a density enormously larger than round either of the hospitals—occurred centred round points which were over a mile away from any small-pox hospital.

At first sight this result is surprising when we consider the enormous differences in density of infection shown in Dr. Reece's table, comparing the circum-hospital areas with the rest of Liverpool. This discrepancy arises from the fact that the density of infection over the whole of Liverpool, excluding the hospital areas, cannot legitimately, in my opinion, be compared with the density of infection over

* It is perhaps scarcely necessary to repeat that the figures in the above Table, for which corrections for house-density cannot be made, are only used like spot-maps as a preliminary test.

any of the special areas; because, as a matter of fact, and as the spot maps clearly show, the infection did not at any time attack more than a relatively small part of Liverpool. The comparison instituted in Dr. Reece's figures is thus between districts which admittedly were at substantial risk from the infection, and districts which were at little or no risk. For the densities of infection of different areas to be comparable, it is obvious that whatever may be the intensity of the infection at work in each area, as measured by its absolute density in that area, the infection must in each case be so distributed that the maximum deviation of density in any element of one area from the mean density over the whole area is the same as the corresponding maximum deviation in the other area. A reference to Dr. Reece's spot maps will show that this test, or any test which does not propose to include districts which were free from infection in the areas chosen for comparison with the hospital areas, will exclude so much of Liverpool from the area with which comparison can properly be made as to deprive the figures relating to the "rest of Liverpool" of any sort of significance in the comparison.

I may point out in passing a somewhat similar—though less wholesale—source of error. This occurs when the density of infection in a small area into which a highly-infectious contagium is introduced is compared with that in a larger area. In Purfleet, for instance, the introduction of a few cases, particularly if "missed," would naturally cause a sufficient number of secondary cases to make a formidable density of infection when calculated on the minute population of the village; in the same way as one or two "missed" cases in a common lodging-house in a large town may determine an outbreak of as many dozen cases, which, if reckoned on the population of the immediate environment, would give a wholly misleading picture of the morbidity of the entire town, or even of one of its wards.

I am bound, therefore, to submit to you as the result of the considerations which I have outlined, that the positive evidence derived from recent experience in support of the hypothesis of distal convection adds little, if anything, to the strength of the case so brilliantly advanced by Mr. Power. And when account is taken of the increased facilities given by compulsory notification, as well as by the mere weight of years, this result can scarcely be interpreted otherwise than as a distinct weakening of the probability that the hypothesis is true.

I am not prepared to urge that a method of convection

which, it is suggested, requires special adjuvant circumstances should appear in every epidemic; but the high proportion of epidemics which have failed to show evidence of aerial convection is a fact of unquestionable significance. When one recollects the considerable periods over which small-pox epidemics commonly last, and the great variety of meteorological and other conditions which occur during this period, the number of negative cases seems wholly disproportionate to what would be expected were the hypothesis correct. My difficulty is in nowise diminished by Dr. Buchanan's observations on this point.

No valid comparison can be made between the failure of a high percentage of small-pox epidemics to disclose evidence of aerial convection, and the fact that in a scarlet-fever epidemic due to milk or some other single infection, perhaps only 10 out of every 100 persons are infected. The 90 persons in the single epidemic escape by chance, or through insusceptibility, or for some other reason; and as these circumstances will probably appear in different combinations in different individuals, the infection attacks a widely heterogeneous body, and will naturally give widely varying results. In a group of 100 epidemics, on the other hand, we have by hypothesis 100 suitable populations, of which the susceptibility and fitness for the disease are shown by the occurrence of the epidemic. The units are not individuals but populations, and in the absence of overwhelming evidence to the contrary, they must be regarded as—and almost certainly are—homogeneous. The variations which occur in this case cannot, therefore, be explained by assuming individual variation among these units. They can only be explained, consistently with the hypothesis of aerial convection, by assuming that in the percentage of failures the necessary combination of meteorological conditions has been absent throughout all those epidemics which have failed to conform to the hypothesis. Whether this assumption is justified or not is a matter on which no great experience of the British climate is required for forming an opinion; but it is a totally different assumption to that on which an infection would be expected to attack all or none of the individuals of a community; and I think that the evidence in favour of the latter needs considerable amplification before the assumption can safely be made.

DR. NIVEN: I shall endeavour to go very straight to the point in what I say. The influence of small-pox hospitals

in producing an increase of small-pox around the hospital appears to me beyond the need of proof; and I believe that the small-pox hospital placed in the midst of dwellings does, in the absence of special precautions such as I shall briefly mention, cause an increase of small-pox in its neighbourhood. It is no answer to take a centre, perhaps in some area of diffusion, place your circles around that, and so obtain artificially-graded figures; that is mere juggling—it really will not in any way meet the question.

In 1893, however, as it happens, I did apply a test, which may be considered a fairly just one, to a diffusion of small-pox which occurred around the Westhulme Hospital, Oldham. I took—not an accidental centre such as I have just referred to, but a common lodging-house in the centre of the town, and drew quarter-mile circles round this common lodging-house. I also took the small-pox hospital as the centre of another set of quarter-mile circles. The percentage of houses invaded by small-pox in an untraced case during 1892 and up to March, 1903, was:—

Within $\frac{1}{4}$ mile of the hospital	...	...	4.7
" $\frac{1}{4}$ to $\frac{1}{2}$ mile "	...	...	1.5
" $\frac{1}{2}$ to $\frac{3}{4}$ mile "	...	...	0.45
" $\frac{3}{4}$ to 1 mile "	...	...	0.42

Round the common lodging-houses, the percentage of houses invaded by untraced cases for the same period was:—

Within $\frac{1}{4}$ mile of the lodging-house	...	...	.42
" $\frac{1}{4}$ to $\frac{1}{2}$ mile "	...	...	.43
" $\frac{1}{2}$ to $\frac{3}{4}$ mile "	...	...	.53
And from $\frac{3}{4}$ to 1 mile "	...	...	.22

The same relation in the incidences round the hospital and round this centre held in the previous outburst of 1888.

I consider that to be a fair comparison, and it is obvious that, taking the part of the town which would be most likely to be affected by small-pox, you get no such graduated increase on approaching this point as you get around the hospital.

I suppose no one would seriously dispute that small-pox hospitals do cause small-pox around them. I think it is necessary to have some experience of the working of small-pox hospitals in order to form a just judgment on that question. I should like very briefly to state precisely what happened in Oldham in 1893, there being then a number of acute cases in the hospital. There were two bursts of

untraced cases which occurred, one about January 9th, 10th, and 11th, and the other from January 29th to February 8th. The first burst comprised six cases, and the second burst 21 cases. A fortnight before the first occasion the wind was variable, but embraced the district affected by the cases; on the second occasion, however, it blew steadily for some days, a fortnight before the outburst, towards the quarter of the town which was particularly affected. All these cases were carefully investigated, and could not be traced to previous cases. Now, I should just like to say a word or two on this subject. In speaking of the different causes to which the cases in the neighbourhood of the hospital may be traced, it has been justly said by Dr. Newsholme that the influence of the hospital, in so far as carelessness of the staff was concerned, would produce precisely the same effect on the graduation of incidence around the hospital as a diffusion of small-pox infection through the air. That is quite true if the movements of the staff are capable of producing the effect: I do not believe that they are. We find numerous contacts from cases of small-pox going to work, and no infection appears to result. It is incredible that the staff of the hospital should produce effects which are not produced by contacts from private houses. These contacts with private cases are subjected to precisely the same kind of infection as the members of the staff; their garments are probably more infected than those of the latter, especially as the staff are obliged to change their clothes before leaving the hospital. Nevertheless, it is rare that you get any cases following the visits of these contacts to works. Although we cannot absolutely exclude contacts as a cause of small-pox, it is so small that it may be disregarded in this connection, and I do not think we can seriously think of that as a possible cause of small-pox outbursts around the hospitals. I should like to press this point a little further. Had small-pox been carried by the staff to cases outside, it is not to be believed that the sanitary inspectors would have failed to produce evidence of contact between members of the staff and at least one case. Yet no such evidence is forthcoming.

Now Dr. Newsholme says that you cannot get an absolute demonstration of aerial diffusion from a small-pox hospital; but I think that what I am about to show you comes as near to being an absolute demonstration as it is possible to get in matters of this description.

I have here a series of maps showing, as from the

1st of January, 1894, the incidence of small-pox in Manchester week by week, from which it is seen that there were three cases—perhaps none, perhaps one, two, three, and so on—down to April, and then they begin to get a little more frequent; very few of them indeed, however, occurring in the district where the hospital was situated. Some of them undoubtedly did; and I do not see how you are to exclude these as not having been caused by the hospital. I will read a few particulars, and then show the maps.

Week ending	April 7th	...	3	cases in	the town.
"	" 14th	...	6	"	"
"	" 21st	...	5	"	"
"	" 28th	...	16	"	"
"	May 5th	...	4	"	"
"	" 12th	...	6	"	"
"	" 19th	...	2	"	"
"	" 26th	...	5	"	"

And then this happened. (In the week ending June 2nd, there were 39 cases, 19 of which were scattered round the hospital.) Here is the Small-pox Hospital (indicated on map), and here are the various fever pavilions. The fever hospital is separated from the adjoining district of Harpurhey by a brook, which was practically never crossed, over the greater part of its course. There is but little commerce in this direction. The wind on the 11th, 12th, 13th, and 14th May was variable, and probably boxed the compass. (The direction and force are taken only at 9 o'clock in the morning.) On the other hand, it was blowing on some days under investigation in this direction, and this, and this. You will perceive that, coincident with these outbursts all round the hospital, you have these five cases occurring in four wards, widely separated, three in the scarlet fever wards, and one in the enteric ward. Now, the two sides of the hospital—the fever and small-pox—were kept absolutely separate, except for the visits of the medical superintendent and matron, who went first into the fever wards. No small-pox had occurred in 1894 in the fever wards previous to these outbursts, so far as I can ascertain; and here you have all these cases occurring at one time, and coincident with the outbursts around the hospital outside, part of which external outburst lies external to, but on lines connecting, the small-pox hospital and the fever wards. The five internal cases occurred, two on the 26th, two on the 28th, and one on the 30th May. In the district round the hospital there occurred on three days, May 26th, 27th, and 28th, fifteen fresh cases of small-

pox, only one of which could be traced to a previous case. Of these fifteen persons, six were housewives, three others were women at home, one aged seventy-three, one a dressmaker, and the third a newsagent. Only five of the fifteen were males. The houses in which they occurred were, many of them, quite out of the beaten track, and away from the approaches to the hospital. The staff of the hospital would generally come down the Oldham Road (in this direction). They might cross the fields, and pass down (in this other direction), but there is no reason to suppose that any of them had small-pox; and it is altogether out of my experience that persons not suffering from small-pox should cause any appreciable amount of small-pox.

Now, you have to afford an explanation of all this, and I say that absolutely no explanation can be given unless you accept aerial diffusion, which is as near as possible to an exact explanation as you can have.

A MEMBER: How near were those wards to one another?

DR. NIVEN: I am afraid I cannot tell exactly, but I can give you a map.

A MEMBER: Can you tell us within a quarter of a mile?

DR. NIVEN: Oh, they were quite close; the grounds being simply separated by a hoarding. The distance of the invaded wards from the small-pox hospital would be about 500 ft. If you consider that you have two such coincident occurrences, you have to find some cause for these cases outside which will also explain the simultaneous cases inside, and I submit that absolutely nothing will account for such an occurence as this except the carriage of small-pox in the air. Of course, if you could explain these cases by direct infection, it would be another matter, though, even then, the double occurrence outside and inside the hospital walls would cast doubt on any history of direct personal infection cases. As I have said already, I think that the occurrence at Monsall Hospital in 1894 is as near a positive demonstration as it is ever possible to get on such a matter. Personally, I should regard my experience as conclusive for practical purposes.

I came to the same conclusion with regard to the outburst of small-pox round the small-pox hospital at Oldham, which occured in 1893. I had had previous experience of diffusion in 1888, when Westhulme Hospital was accused of radiating small-pox, and in my capacity of Medical Officer of Health I repudiated that view. I went to every

house in which small-pox had occurred, and I certainly picked a good many holes in the statement which had been put forward, so much so that the Local Government Board considered the charge "not proven." But although that was so, there remained a doubt upon my mind whether, taking all the facts into consideration, these cases might not after all have been due to the hospital. Then, when we got such a sweep of small-pox as that occurring in Oldham in 1893, which we could not trace to previous infection, or find any explanation of, I felt that I must give up the battle. We, therefore, moved all the recent and fresh cases out into the country, still retaining Westhulme for convalescents. The result was that there was practically no small-pox around Westhulme Hospital in 1894. The same thing happened in Manchester in 1894. We moved all the acute cases from Monsall, still using Monsall for convalescents. The cases then ceased to occur in the district of Newton Heath, in which Monsall Hospital is situated.

I had previously been persuaded—perhaps too easily—that patients could not conveniently be oiled: that it made them sick, and that the other inconveniences were very serious. But I considered that, as we had to treat the acute cases at Clayton, we ought not to run any risk of having small-pox diffused; and from the beginning of treatment of acute cases there, in 1894, we had all the patients in the acute stage oiled daily. For the last two or three years they have been oiled twice daily. I have recently been making inquiries as to this practice, and I think perhaps I may have laid too much stress upon the difficulties. I am told that the patients who are not too seriously ill oil themselves, and that they like it; in fact, they find the irritation of the skin reduced by the 2½ per cent. carbolic oil. Of course, oiling of the very bad cases is rather unpleasant for the nurses. I have no doubt that the disease has been prevented from spreading to the houses in the vicinity by this means, and I have also no doubt that the same mode of treatment would again suffice to prevent the diffusion of small-pox. It is, at all events, a refuge for people in difficulties, who cannot get a hospital sufficiently removed from centres of population.

Dr. Hope: Unfortunately, I was not able to be present when Dr. Buchanan's Paper was read, but he has been good enough to send me a copy, and I have given it very careful study.

I should like to say that nobody doubts the ability of small-pox hospitals in this country to cause the spread of small-pox in their vicinity—there is no question about that, I take it. The question at issue is whether the presence of small-pox in the vicinity of hospitals is to be ascribed to aerial convection, or whether it is due to contact with infected persons or things either from the hospital or from other infected sources: whether, in fact, its presence in the vicinity of the hospital is not due to precisely the same causes that give rise to it in parts remote from the hospital.

At the outset of his Paper, Dr. Buchanan mentions the part played by acute cases of small-pox. Now it is to acute cases of small-pox that the mischief is ascribed, and I should like Dr. Buchanan to give us a definition of an "acute case." All small-pox at the outset is acute, as everybody knows; but when modified by vaccination the acute symptoms pass away soon, and the patient becomes convalescent. The risk of infection in these cases is inconsiderable, unless the patient is in close and intimate contact with susceptible persons; in such ways, for example, as living in the same house, sleeping in the same bed, travelling in the same carriage, or working in the same room.

On the other hand, in unmodified cases of small-pox, long after the acute stages have passed by, the patient in hospital is eminently infectious; indeed, the ancient Chinese method of inoculation was by means of powdered scales or scabs from an infected person. So, when patients are transferred from one hospital to another, they may be in an infectious condition or they may not. The term "acute cases" is, therefore, an unfortunate one, and conveys very little meaning.

One point in Dr. Buchanan's Paper—a feature also which is conspicuously noticeable in all his Reports—is the eminent fairness with which he gave consideration, not only to the facts which tend to support his views, but those which appear to have an opposite tendency, There is, in fact, in his official Reports not less recognition and prominence given to the causes of spread of small-pox other than aerial convection, than to those circumstances to which the explanation of aerial convection alone appears to him to be applicable. In this respect he has followed the essentials of scientific investigation, as well as the methods to which the Local Government Board Reports owe so much of their value.

The conclusions to which Dr. Buchanan's experience and investigations have led him are, in one important respect

similar to those of Mr. Power, viz., the importance of the peculiar atmospheric conditions which are likely to be conducive to the distribution aerially of infective matter, and to the fact that these conditions are not uniform nor always active; but that they may, from time to time, so combine as to render the transference of particulate infective material as easy as the transference of dust.

With the other outbreaks to which the writer of the Paper refers, viz., those at Orsett, Glasgow, and Manchester, I do not propose to deal, as the short time at my disposal will be fully occupied with the outbreak at Liverpool, and to the Report of Dr. Reece upon it, which is so largely drawn from in Dr. Buchanan's Paper. That Report, upon the face of it, would almost convince any person unfamiliar with the subject, that the Liverpool hospitals had proved a grave source of public danger.

In commenting upon Dr. Reece's Report, I, of course, confine my remarks to those aspects of it to which the writer of the Paper has alluded. There are other features in that Report which, in due course, and in the proper place, will be dealt with.

One of the most remarkable features in connection with Dr. Reece's Report is, that from start to finish there is no reference whatever to any single one of the considerations upon which the Reports of Mr. Power and others, and of Dr. Buchanan himself, laid so much emphasis. There is no suggestion of any other possible cause of the spread of small-pox except aerial convection; and this he assumes to take place without any of the meteorological conditions upon which Dr. Buchanan lays so much stress.

Mr. Power was careful to point out that—

"Small-pox is a disease infectious beyond all others of its class. Not only does it spread with greater facility than, for instance, scarlatina or typhus, but the measures of isolation and other precautions against dissemination which suffice with those diseases are, as regards small-pox, altogether futile."

He then goes on to speak of the spread of small-pox by means of bedding, etc. He then adds:—

"Cases of small-pox, themselves so little serious as to be mistaken for 'chicken pox,' have in our ordinary experience the power of producing in unprotected persons severe attacks of the disease And other slight cases of small-pox, not mistaken but purposely concealed, do much in all experience to spread the disease in an epidemic form."

Clearly, in investigating the causes and progress of the extension of an outbreak of small-pox, no circumstance should be rejected which has an obvious and distinct bearing upon the outbreak; and in preparing a statement

for the judgment of others, it is well, in matters of doubt, to state the facts on both sides of the question.

I would criticise Dr. Buchanan's Paper so far as it rests upon this report of Dr. Reece's.

I would point out that Dr. Reece, before entering upon his investigation at all, states distinctly that there is only one explanation of small-pox incidence around hospitals, viz., dissemination of infection by aerial convection, and that that explanation is absolutely satisfactory to him. This is his substitute for the temperate and extended view of the question expressed by other writers. So reluctant is he to disturb his satisfaction, that he rejects all the considerations upon which so much stress is laid by Mr. Power and other observers, and makes no reference whatever to the volume of proof laid before him as to other definite sources of infection.

It is clear that Dr. Reece bases his case against Liverpool and his conclusions, upon this assumption and upon maps, carefully prepared, the accuracy of which I fully admit. So confident is he that this view comprehends the entire situation, that no other aspect of it is deemed worthy of consideration. The question may, in the first place, be considered from Dr. Reece's standpoint, and the further evidence considered subsequently.

In the city of Liverpool there are three other hospitals, besides those under discussion, used for the various infectious diseases other than small-pox. I have taken one of these, viz., the Netherfield Road Hospital, and put rings round it in the same manner as Dr. Reece has done round the small-pox hospitals. The concentric zones have been placed round it, and the map has been prepared by the same people who prepared Dr. Reece's maps. And what we find is as described in Tables on opposite page.

These are the zones which have suffered all through the outbreak, and they show more marked gradation than in the case of any of the other hospitals. What would have been said if we had used that hospital for small-pox cases? We might have done so, and if we had, this would undoubtedly have been held up as conclusive evidence—evidence beyond question—of the results of aerial convection. But these cases, which occurred in the neighbourhood of Netherfield Road Hospital, were, in a very large proportion of cases, clearly traceable to other sources, which I have defined and described elsewhere. It has also been proved upon similar evidence, and in the same way, and in an equally large proportion of cases, that the presence of

TABLE showing per 10,000 Houses, and for the period December 8th, 1901, to November 9th, 1903, Houses invaded by small-pox in areas respectively within one mile, within three-quarters of a mile, within half a mile, and within quarter of a mile of the Hospital at Netherfield Road (which did not receive cases of small-pox). The corresponding rate far the rest of the city (including the three hospital areas of the Parkhill, Fazakerley, and Priory Road Hospitals) is given for comparison :—

Period of the Outbreak of Small-pox in the City of Liverpool.	Netherfield Road Hospital Area : House Invasion per 10,000 Houses, within—				House Invasion per 10,000 Houses in the rest of the City of Liverpool, including the Hospital Areas of Parkhill, Fazakerley, and Priory Road.
	1 Mile of the Hospital.	¾ Mile of the Hospital.	½ Mile of the Hospital.	¼ Mile of the Hospital.	
Dec. 8th, 1901, to Nov. 9th, 1903 (23 months).	163.8	176.7	199.8	311.7	96.1

Netherfield Road Hospital (which was not used for Small-pox). Houses invaded per 10,000 Houses in each Zone.

Zones.	0 to ¼ Mile Houses in the Zone, 4,716.	¼ to ½ Mile Houses in the Zone, 11,944.	½ to ¾ Mile Houses in the Zone, 17,692.	¾ to 1 Mile Houses in the Zone, 14,354.	The rest of the City.
Invaded houses .	311.7	155.7	154.8.	133.0.	96.1

small-pox in the vicinity of the small-pox hospitals had a precisely similar origin; whilst there is no shadow of proof to connect any of them with any other source of infection.

Information regarding small-pox is more easily obtainable than formerly, and especially so in Liverpool. There has been important and special legislation in recent years, and this legislation is not designed merely to supply information, but to aid sanitary authorities in safeguarding the public in the prevention of disease, We have, of course, the Notification Act, which is applicable throughout the country. We have also chicken-pox scheduled as a compulsorily-notifiable disease: and this is a very important thing, because in the recent outbreak fifty-four persons supposed to be suffering from chicken-pox, and who were being treated for that disease, were found to be suffering from small-pox.

But even this legislation is not sufficient to meet all the necessities of the case. Many inquiries have to be made, and it is not uncommon for relatives and friends to give untrue replies to those inquiries. Hence it was that the Liverpool Corporation, in 1902, obtained Parliamentary powers to deal with persons guilty of wilful suppression of the truth, or of giving false information with regard to small-pox; and they obtained important penal clauses, which dealt sufficiently with these offences.

The valuable assistance in tracing disease and verifying the sources of infection which each and all of the foregoing Acts of Parliament now afford, were, of course, not available until the respective dates at which they were passed; but they were all, especially the 1902 clause, of great assistance in tracing sources of infection during the outbreak we are considering. But there is no reference to these aids to investigation, nor to their important results, in Dr. Reece's Paper, although they were placed before him. I shall, therefore, ask for five minutes' indulgence to supplement the omission; but I wish first to refer very briefly to the hospitals themselves.

Taking them in the order in which they have been dealt with in the Paper:—

The Fazakerley Hospital is a new hospital for small-pox, erected within the last few years by the Liverpool Corporation upon a site which was very carefully considered. The Corporation were desirous to place their hospitals in such a position that they should not be encroached upon by buildings in the future, and that patients should have such aids to recovery as fresh air would afford them. The amount paid for the site, which consists of 130 acres, was close upon £40,000. The Corporation were aided in their selection of a site by an experienced inspector of the Local Government Board, who, in 1898, gave it his warm approval. Subsequent to that, a small additional quantity of adjoining land came into the market; and in order to borrow the money for its purchase, as well as to borrow £20,000 for the hospital itself, another medical inspector of the Local Government Board, of great experience, investigated the subject, and he also expressed his approval of the site in question. Finally, a third application for more money for hospital purposes was made, and a third inspector of the Local Government Board, after investigation, approved of the site. It will be seen, therefore, that the site was the best which foresight could suggest, and which money could procure. Until Dr. Reece's report

appeared, there was no suggestion that this institution had been other than a benefit to the city. It might be, of course, that the three inspectors of the Local Government Board, as well as the medical advisers of the Corporation, are wholly and entirely wrong, and that Dr. Reece is right; but it cannot be expected that his views will be accepted without very rigid inquiry. Clearly, there was more at stake than the mere reputations of those who gave an opinion in favour of the site. If it were unsuitable, there can be but few sites in the kingdom which are suitable for small-pox hospitals at all, for not many Corporations would be in a position to pay so large a sum as £40,000 for a site.

According to Dr. Reece's Table, on page 14 of his Report, the house-to-house invasion within a quarter to half-a-mile zone of the Fazakerley Hospital, was four times as great relative to the city invasion as it was in the quarter to half-mile zone at Priory Road Hospital. What is still more remarkable is that the house invasion in the half- to three-quarter-mile zone at Fazakerley is, according to Dr. Reece's Table, eighteen times as great relative to the city invasion as it is in the corresponding zone of the Priory Road Hospital; further, in the three-quarter to mile zone it is twelve times as great. Yet the Priory Road Hospital falls short of modern requirements: it is not one which comes up to our standards, and is, moreover, in a situation altogether unusual in regard to the population around it. Priory Road Hospital, nevertheless, is less dangerous than Fazakerley Hospital.

It must be mentioned that Fazakerley Hospital has only got nine houses within the quarter-mile zone, and a total of one hundred and seventy-five houses within a half-mile zone of the hospital. Similarly, the number of houses within a mile is relatively very trifling, as compared with the other two hospitals. There were only two houses invaded within the entire half-mile circle, and it is a wilful misuse of figures to prepare Tables which suggest an enormous house invasion upon facts such as these.

Furthermore, the great majority of the few cases within the mile zone occurred, not at the time when the hospital was at its fullest use, but on the contrary at a time when the hospital was least used; whilst they dwindled away to a minimum at a time when the hospital was at its fullest use.

I notice that Dr. Buchanan (very wisely and properly) attaches so little importance to Dr. Reece's Table relating to Fazakerley, as to take no notice of it. I do not know whether this is out of regard for the appreciation which his colleagues have for the site; but it is a wise course,

for, upon reasons which will shortly be more apparent the Table is valueless and misleading, and it is appropriately discarded. I do not say this because a large sum of money was spent upon the site, or because the site was approved of by others besides myself. All of us may have been absolutely mistaken; and, if so, the Corporation would at once cease to use the hospital, and, with the sanction of the Local Government Board, buy another site more suitable, the first object being, of course, to advance the public health and the public safety.

But Dr. Buchanan assigns no reason for his rejection of the Table relative to the Fazakerley Hospital, and his acceptance of those relating to the other two hospitals.

With regard to Priory Road Hospital, which it is alleged has done so much damage, there is one other striking fact, and that is: that the population living within a quarter-mile of it, though not large, is about three times the number which has been regarded as the margin of safety; yet this population of five hundred persons of all conditions, living within the quarter-mile zone of the hospital, has, throughout the entire eighteen months that the hospital was used for acute cases of small-pox, been absolutely free: only one case having appeared within the quarter-mile zone throughout the whole of the period.

Now turn to Park Hill Hospital. Figures are given suggesting an enormously greater incidence on the quarter-mile radius of this hospital than the other one, or, indeed, anywhere else; namely, 526 per 10,000, as against 85 for the rest of the city, excluding the hospital areas. Now 526 is a very large figure to use, and it is a relief to know that it means the invasion of only nine houses during the time the hospital was in use. But prior to the use of the Park Hill Hospital for the treatment of small-pox, the disease had repeatedly been present in its vicinity: in January, 1902, a house within the three-quarter-mile zone was invaded by small-pox; in February, 1902, one house was invaded within the quarter-mile zone, and two houses within the half to three-quarter-mile zone; in March, 1902, a house within the half to three-quarter-mile zone was invaded, and one between the three-quarter and mile zone; in November, 1902, one house in the three-quarter to mile zone was invaded; in January, 1903, three houses were invaded within the half and three-quarter-mile zone, and one house between the three-quarter and mile zone. So that there were twelve separate and distinct invasions of

houses within the alleged zone of influence before the hospital received its first case of small-pox.

During the use of the Park Hill Hospital for small-pox, nine houses were invaded within the quarter-mile zone of the hospital. These nine houses are represented by the figure 526.3, quoted in Dr. Reece's Table, as the instance in which the influence of the hospital is most pronounced. Now, what probability is there of these nine invasions having an origin analogous in character to the preceding twelve: which occurred in the vicinity before this hospital was used for small-pox at all? And if any of them should be proved to be infected in that way, what becomes of the Table and the inferences drawn from it? Four of the nine patients had, it was known, been exposed to infection elsewhere—did they get their infection from this source, or did they get it from the hospital? I will not waste time by quoting these cases: they are in print, and they will be duly put before the Society.

Let us now turn for a few moments to the study of the actual proofs of the sources of infection, both within and without the various zones; for, believe me, the behaviour of small-pox was absolutely identical in both. The Acts of Parliament which I have quoted, in the hands of a staff of adequate size, training, and intelligence, proved what was the actual source of infection in about 1,000 out of 2,082 cases; and out of these 490 were actually found by watching those who were known to have been exposed to infection, before any medical man had been called in. The staff was very much augmented for these purposes. Any person who had been exposed to infection in the centre of the city or elsewhere was kept under observation, and if he exhibited any symptoms he was noticed at once and visited medically. In addition to these, and to the 54 cases which were treated as chicken-pox, a large number of mild and unrecognised cases had been roaming about town within and without the zones, whose illness was only recognised by the fact that they infected members of their families or friends. Were these aerially infected from the hospital? Their infection has been ascribed to it, notwithstanding that their source of infection was the same as that of patients living miles away from the hospital.

I should like to quote from a Report on this particular matter, which I published in 1903:—

"Doubt as to the nature of the illness proved another fruitful source of dissemination, owing to the fact that a large number of cases, modified by vaccination, were regarded by the medical attendant as chicken-pox; it

became necessary to schedule chicken-pox as a notifiable disease, in order that doubtful cases might be visited by a medical officer experienced in small-pox. The value of this measure is confirmed by the fact that fifty-four cases of small-pox, which were under treatment as chicken-pox, were discovered and dealt with. Unfortunately, however, there were still many cases of so mild a type that no medical man was called in; and it was only after the severe infection of others by these cases that the real nature of the disease was evident. A further mischief connected with this mild form of the disease arose from the patients going in public conveyances to places of public resort, or to places of business, laundries, tobacco works, tailors' shops, etc., not only infecting those with whom they associated, but infecting articles which they handled. Children and other inmates of their houses were also going to and fro to their daily duties. In several cases the patient, in doubt as to the nature of his illness, had sought advice at the out-patient department of the charitable medical institutions, and had sat amongst the crowded occupants of the waiting-room until his turn came to see the doctor. Re-introductions of the disease were frequent, by tramps, labourers, and so on."

I should also like to quote the case of Messrs. G. W. & Co., six of whose employées fell ill at the same time at their homes in different parts of the city, pointing clearly to one infection and at the same time. Of these six cases, one lived within the zone of Priory Road Hospital, one in the zone of Fazakerley Hospital and one of Park Hill Hospital, the remaining three in parts all outside the hospital zones, whilst they all worked in the centre of the city. Now, what was the source of infection of these men? Was it that aerial infection from three different hospitals picked out three of them, and was, in fact, the cause of their illness, whilst the infection of the other three was from other sources?

I will just mention two other cases, and quote no more at present. At Everton Brow a servant-girl in a public-house was affected with small-pox so slightly that it was at first unrecognised. The nature of her illness was verified when the manager and his daughter had been infected. Her relatives were infected in another house, and they in turn passed on the infection to fellow-workmen residing severally in three or four other different parts of the city, all of which were outside the mile zone of any hospital excepting one, which is within the mile zone. Now, that one is said to be due to aerial convection from the hospital. If it is not, what becomes of the maps and dots? There is no reference to any other causes; that is what one—I will not say complains of—but surely all this evidence ought not to have been suppressed altogether.

Henry S. Morris and his father, of No. 83, Admiral Street, were being treated as cases of chicken-pox.

On March 7th, 1902, the doctor communicated with the Medical Officer of Health that he had suspicions regarding

the illness, and the Assistant Medical Officer of Health forthwith visited the patients, found them to be suffering from small-pox, and had them removed to hospital.

A relative of the above persons, named Annie Robinson, living at No. 14. Balkan Street (which is within fifty yards of Parkhill Hospital walls), was notified by a doctor on March 7th to be suffering from small-pox. She had been ill since February 27th, and had been in the habit of visiting her relatives at No. 83, Admiral Street, where small-pox existed. The dates of illness confirmed this source of infection.

There were no cases of small-pox in Parkhill Hospital at this time.

Now, Dr. Reece's attention has been called to whole series of such instances; instances, in fact, so numerous that, if he were willing to admit the possibility of such infection, the whole of his spot-maps would be wasted labour. These facts have been brought under his notice; but he prefers to abide by his conclusion that all these cases are due to aerial convection: thus claiming for aerial convection selective qualities which nobody else has claimed for it, and for which no explanation is offered. Dr. Reece may disclaim this view; but if he disclaims it he must modify his spot-maps, and if he modifies his spot-maps, he acknowledges that his labour has been in vain, and his conclusions fall to the ground.

There is only one other point. You will recollect what has been said about the situation of the Fazakerley Hospital, and these maps and charts show you plainly enough. If you look at the maps, you will find that there was the maximum number of invaded houses in the vicinity at the end of about the forty-eighth fortnight, at a time of minimum use of the hospital, whereas the maximum use of the hospital was made about the twenty-seventh to the thirty-first fortnight, when there was a minimum of house invasion. There can be no question of incubation that can spread over all that time.

I should like to say just one word with regard to the main question treated in Dr. Buchanan's Paper. I do not suggest for a moment that Dr. Buchanan's conclusions are not correct. But he weakens his case by bringing in instances of which he himself has no knowledge, and which depend altogether on dots and maps, and the sweeping aside of every other consideration. If an investigation into the causes of accidents were ever made, and the investigator contented himself by going to a hospital for

accidents, drawing rings round that hospital and putting dots for the accidents, and then drawing the conclusion that all these accidents are to be ascribed to the influence of the hospital, his conclusions would be obviously wrong.

DR. BUCHANAN: I should like, Sir, to ask Dr. Hope: Can he say if there is anything in Dr. Reece's Report—and, if so, where—which gave him the impression that Dr. Reece stated that all these cases were due to aerial convection? I have studied the Report from cover to cover, and I do not find anything in it which leads me to suppose that he says that every dot that appears in the maps is a case due to aerial convection.

DR. HOPE: He does not say so in so many words; but his conclusions point to that conclusively from the fact that he excludes all other possible sources than aerial convection. In your own Reports we find due prominence given to other sources of infection, and due allowance made for them. In Dr. Reece's Reports there are not any. If he were willing to admit that patients within a mile of the hospitals were infected through the usual channels, his conclusions would fall to the ground.

DR. MCVAIL: I have read Dr. Buchanan's Paper with very great interest, but would not have troubled coming from Glasgow to hear it, because I so heartily concur with his conclusions. But when Dr. Newsholme agreed to open the discussion on the other side, I was anxious to learn what could be said against aerial convection. I have heard the theory denied, sniffed at, called ridiculous; but none of these attacks satisfied me as sound argument. I was therefore very glad to have the opportunity of hearing Dr. Newsholme, who is always able to say everything that can be said in support of his case.

It is very pleasant to learn two things as to Dr. Newsholme's position. First of all, that he is merely an agnostic, taking no side for or against aerial convection. He is not convinced—he does not deny the theory, but he simply does not know.

Secondly, he thinks it of no practical consequence whether aerial convection occurs or not. He agrees that small-pox hospitals are centres of infection, and consequently that such hospitals should be situated at a distance from populous places. If that is the general feeling throughout the public health service, there must have been

a greater advance of opinion than I have been aware of. It was stated, not long ago, by a highly respected authority, that aerialists are the opponents of hospitals for small-pox; but it appears now that non-aerialists, or agnostics, are opponents of small-pox hospitals in exactly the same sense as aerialists—both holding that these hospitals should not be situated in populous places.

I did not clearly make out whether Dr. Newsholme is of opinion that these Liverpool hospitals, into whose figures he went so minutely, had any relation to the small-pox around them; whether the fact that when the one hospital only was in use, that hospital became surrounded by small-pox, and the subsequent fact that when all three hospitals were in use, all three became surrounded by small-pox; whether Dr. Newsholme came to the conclusion that these were mere coincidences, and that there was no causal connection at all.

DR. NEWSHOLME: Mr. President, I must leave the general trend of my remarks as I have already made them. I may simply say that my own opinion is, that there was no proof of hospital influence.

DR. MCVAIL: Dr. Newsholme is of opinion that quite possibly neither aerial convection nor intercourse with the hospital had anything to do with the excessive prevalence of small-pox: first of all, around the one hospital when it was alone in use; and, secondly, around the three hospitals when they were all in use simultaneously.

DR. NEWSHOLME: I believe that small-pox is always liable to be spread by intercourse between the hospital and its surroundings; and if there was such intercourse, that may have increased the prevalence of small-pox in the neighbourhood. Whether there was such intercourse or not I cannot say.

DR. MCVAIL: At the Congress of the Sanitary Institute in Glasgow—at a meeting at which I was not present—I noticed afterwards that an opinion had been expressed, to the effect that the decision of Mr. Justice Farwell in the Nottingham case had settled the question of aerial convection. That view seems to have got some hold, and, as I have said elsewhere, it is therefore desirable to note what really appears to be the relation of the Nottingham case to the theory of aerial convection.

Mr. Justice Farwell—as reported in the medical press—

stated that, in order to obtain an injunction in such a case, it was necessary to establish "a strong probability, almost amounting to a moral certainty," of risk or danger following from the hospital. He stated that to be the law of England, and it is also the law of Ireland, for he was quoting from the opinion of an Irish judge. That being the law, the decision in the Nottingham case could not be other than as given by Mr. Justice Farwell. No one who appeared in that case—and perhaps no one who supports the theory—would make the statement that, with regard to every such small-pox hospital there is "a strong probability, almost amounting to moral certainty," that small-pox will spread from it. The view of the aerialist—as I understand it—is, that it requires a concurrence of favourable conditions to produce this result, and that concurrence cannot be expected with anything approaching to certainty. Mr. Justice Farwell's decision does not in any way invalidate the theory of aerial convection. He expounded an important point in the law of England, but the state of the law does not affect the medical theory.

But Mr. Justice Farwell went on to give his legal view as to the nature of the evidence which would be required in a matter of the sort, and that view is of the very greatest interest. He held, with reference to Dr. Thresh's evidence, that "the plaintiff's case depended on the inference to be drawn from an unbroken series of facts—in all cases where A has occurred B has followed, therefore A causes B. But the conclusion depends on the universality of the premiss, and a negative instance unexplained spoils the chain."

Now, does that formula apply in the field of medical science, where we deal not with abstract problems in which the factors are assumed to be unchanging and unchangeable, but with a variable and as yet undiscovered micro-organism, conveyed by a variable medium to a variable human subject? Suppose we have a case of scarlet fever which is not followed by a second case, and that there is no explanation of the failure to infect. It appears, then, that because B did not follow A, scarlet fever is not, at common law, to be regarded as an infectious disease. Fortunately, disease prevention is carried on under statute law, not under common law.

In September, 1902, in *Public Health*, I read this:—

"Two cases of virulent small-pox occurred in a crowded slum neighbourhood, and were not removed to hospital; and, although the neighbourhood included a number of unvaccinated persons, no spread of infection occurred."

Here there was opportunity of infection by contact; but B did not follow A; and if "a negative instance unexplained spoils the chain," then small-pox cannot be legally regarded as infectious by personal intercourse, any more than by aerial convection. I do think that the Nottingham decision does not take us very much further than we were before.

What attitude should be adopted with regard to evidence on the question of aerial convection? If the attitude be this—that aerial convection can be admitted only when the possibility of all other sources of infection is absolutely excluded—then there comes the further point: Is it not also held to be invariably impossible to exclude absolutely all other such sources? If it be required of believers in aerial convection that they shall prove a negative (namely, that no other possible cause of infection did exist), at the same time that it is pointed out to them that a negative is unproveable, obviously the requirement cannot be fulfilled. Surely, the same rules of evidence should be applied to aerial convection as to other questions in the domain of medical science—rules, for example, which take cognisance of the fact that we cannot actually see the virus of infectious disease—of scarlet fever, or typhus, or whooping-cough, as it passes from one individual to another, but have to form our opinions on a basis which must fall short of actual demonstration.

I noticed, some time ago, a paper on this subject by a very competent medical man. He had to do with a small-pox hospital, and he said that he treated small-pox in the same hospital with other diseases; but that the measures that he was taking with regard to small-pox were quite sufficient to prevent its spreading, and it had not done so. And then he went on:—"I must also add that if the use of this ward for small-pox is found to affect the other pavilions, I shall be quite satisfied that the regulations have not been carried out." That is an example of a mental attitude which I would venture to deprecate in this discussion.

In *Public Health* for September, 1902, there was a leading article dealing critically with the theory of aerial convection. In the course of that article this passage appears:—

"Another experiment has been tried in London at the South Wharf Shelters, to which at least eight thousand cases have been conveyed *en route* for the hospital ships. The shelters hold twenty-five patients, and on one occasion as many as 120 patients have been detained there and on the steamboat when the traffic on the river was stopped by fog; and yet, as Dr. T. Orme Dudfield has pointed out, without ill effect on the locality. There has

been no spread of the disease in the neighbourhood of the shelters; and, in fact, their immediate vicinity has been freer from small-pox than many other parts of the borough."

That seems a pretty clear case of absence of aerial diffusion; but, in the January following, this letter appears:—

"In the September issue of *Public Health* a statement was made to the effect that there was immunity from small-pox in the vicinity of South Wharf Shelters; and this is again mentioned by Dr. Malet, in his letter in the December issue. It arose from a statement made by myself to the Medical Officer of Health for Kensington, early in the late epidemic, and when many cases had not occurred in the vicinity of the place in question. Subsequent events, however, proved that this immunity did not continue, and there was a much greater incidence in this region than in any other part of the borough.

"In my special report, published early in October, you will find a full statement of the facts in regard to the Wharf, as far as could be ascertained, under the head of 'Aerial Diffusion.' This statement ought to be corrected, as it is being constantly re-quoted, and is quite erroneous. Taking the area round the Wharf, there are about 2,560 inhabitants cut off from the rest of the borough by docks, with an attack-rate of 10.55 per 1,000 inhabitants, as compared with 5.35 in the next most infected area of the borough, and 2.30 for the whole borough. I have discussed the ways in which the Wharf has influenced this incidence, and I certainly think that danger is to be looked for in the direction of intercommunication between persons employed in the hospital and the surrounding inhabitants, and not through aerial diffusion of the infection."

Thus we see that in September the supposed absence of small-pox is at once accepted and cited as evidence against aerial convection; but the subsequent discovery that small-pox had had a fourfold prevalence in the neighbourhood of the wharves is not admitted to yield any presumption in favour of such convection.

Dr. Louis Parkes, who was in the chair at the Newcastle Meeting of the Royal Sanitary Institute this year, made a very shrewd remark in closing the discussion. He said that he thought all negative cases should be investigated equally with all positive cases. I do think that is a most valuable suggestion. The incidence around small-pox hospitals in populous places should be gone into, and the whole facts stated. It is quite possible that what may appear due to aerial convection at one time may subsequently prove to be in a large measure due to other causes. I think that Dr. Savill did something to indicate that in the case of Warrington. On the other side, there is the supposed negative case of the hospital wharves which I have just mentioned. Also, it has been most fortunate that Dr. Hope's views as to the absence of hospital influence in Liverpool have been traversed by Dr. Reece; and when one gets time to look into Dr. Hope's remarks, it will be possible more thoroughly to weigh up the facts. But I

confess I was surprised at Dr. Hope assuming that the fact that Dr. Reece, in his report, has made no reference to the possibility of other causes of small-pox in the quarter and half-mile circles meant that Dr. Reece denied any such other causes. It is manifest that there must be other causes, but what we want is to explain the *difference* between the larger incidence round hospitals and the smaller incidence in the rest of the community. It is the difference that is in question, Obviously, the existence of a small-pox hospital does not prevent ordinary means of infection around it. That goes without saying.

There was one group of experiences mentioned in the course of the Nottingham case that I have been hoping to know more about. Dr. George Reid gave weighty evidence referring to seventeen hospitals in Staffordshire, from not one of which had there been any evidence of spread of small-pox. It is most important that the whole of the facts regarding these seventeen hospitals should be published. It is impossible for a witness in the box to arrange and elaborate his evidence as he can do in a Paper on the subject; and just as the Liverpool hospitals' experiences can be best studied not solely from Dr. Hope's evidence in the Nottingham case, but also from his Annual Report, read alongside of Dr. Reece's Report to the Local Government Board, and from Dr. Hope's contribution to the present discussion, so one would desire to have Dr. Reid's valuable experience recorded in detail, giving the full facts regarding every one of the seventeen hospitals: the population around them, the times when they were used for small-pox, the numbers of acute cases, and the precautions against personal conveyance of the disease. Then we could weigh the evidence along with Dr. Hope's and Dr. Thresh's, and the Glasgow, Gateshead, Essex and other cases, and consider the whole volume of such evidence at our leisure.

I am quite at a loss to discover where the difficulty lies in accepting the possibility of aerial convection of small-pox from hospitals. We agree that small-pox is conveyable for at least some distance through the air. We agree also that small-pox and scarlet fever and diphtheria may be spread directly or mediately from any case or cases of the disease; and so theoretically all these diseases may be spread by hospital intercourse. But is it not possible that prevalent views of the power of small-pox to spread by intercourse are exaggerated? Already I have referred to two London small-pox cases which caused no spread of infection. At Fulham Hospital, when small-pox prevailed there,

nine non-resident servants did not carry the disease to their houses in nine different streets, but so soon as a hospital dustman himself caught small-pox, he infected his own family. We never see scarlet fever or diphtheria spreading from a hospital as small-pox does. Why, then, should the excessive prevalence of small-pox be always and wholly attributed to intercourse? Why especially should this be so when we know that a hospital like Darenth may contain hundreds of convalescent small-pox patients in all stages of desquamation, with crusts and scabs abundantly shed from their whole integument day after day, and yet without any extension of infection from the hospital; whilst on the other hand hospitals containing acute cases closely confined to bed, and previous to dessication of the pustules, cause prevalence of small-pox all around them? If the cause were intercours or mediate infection from small-pox dust, surely a convalescent hospital should be a prime source of danger, instead of being found practically harmless; and, per contra, hospitals with acute cases confined to bed, and previous to drying of the eruption, should be much less effective as centres of infection. But experience shows that small-pox has, over and over again, spread from hospitals containing a collection of acute cases. These considerations are the very basis of the aerial theory. It is simply an endeavour to explain known facts: to construct a working hypothesis which shall adequately account for the observed phenomena of small-pox hospital influence.

Indeed, I do not myself see why, when there is exceptional prevalence of small-pox round a hospital, every case regarding which even the most far-fetched and improbable suggestion of mediate infection can be made, should at once be dogmatically set down to that imagined cause, rather than to atmospheric diffusion.

The evidence is not confined to this country. Aerial convection was discussed in America before the end of the eighteenth century; and Sir John Rose Cormack, about a quarter of a century ago, noticed small-pox being spread from hospitals in Paris, and formed the opinion that it was due to what he called epithelial drift. He also referred to one reported case, where it was believed to have spread specially from that side of a small-pox hospital which had open windows.

After all, it seems to me that the greatest difficulty in accepting the theory of aerial convection is to be found in the statements that have been repeatedly made regarding

the absence of small-pox in certain public institutions within a quarter and a half mile of small-pox hospitals. That was what concerned me a dozen years ago in looking into the matter, and I am struck by the recurrence of such evidence in the present discussion. I would like, therefore, to say a word regarding it. Last week you had Dr. Ker, from Edinburgh, who gave you some account of what has taken place there. What I understand is this—that there is practically no population, excepting in certain institutions, within half a mile or more of the Edinburgh Hospital.

DR. BUCHANAN: Except a small group of dwellings at this end.

DR. M'VAIL: But if within the half mile there is no general population, I think it is necessary to point out that this was not a control experiment. The nature of a control experiment is this: that the two cases shall resemble each other in every point, excepting the one on which the control experiment is made; that is, we should have within a half-mile of the hospital institutions that escape small-pox, at the same time that we have a surrounding population which does not escape. With regard to Edinburgh, these conditions seem not to have been fulfilled. If there was no population in this radius round the hospital except in the institutions, then one does not know whether there would have been any spread of small-pox if there had been a population. The value, in the meantime, of the Edinburgh case seems simply this: that there was a population within that distance of the hospital which did not take small-pox; but whether that was due to its being an institutional population or not, is not in the least proved. In so far as the patients in the fever hospital were confined to bed, or to the wards, they would be much less exposed to aerial infection than persons moving about outside; and that remark would perhaps apply even to the erysipelas cases who were nearest to the small-pox wards. The case has its own value, but its value is limited; and I confess that those of us who know Edinburgh, and the standing of its medical profession, and how the vaccination laws are obeyed in that city, can have little difficulty in believing that you might have institutions there whose population was practically insusceptible owing to vaccination.

In order that such institutional cases should yield a lesson of any real value, it is necessary to know quite definitely the vaccinal condition of the inmates. When, as has

repeatedly occurred, I have been compelled to send small-pox to an ordinary fever hospital, I have examined the vaccinal conditions of every patient in the hospital; and the number who were found not to require vaccination owing to recency of primary vaccination, or to re-vaccination, or occasionally to previous small-pox, has been surprisingly great. For the rest, I have never failed to persuade those who really needed renewal of protection to submit to the operation. In consequence, the institution has been simply insusceptible to small-pox. It is not enough to know that, under risk of small-pox, re-vaccination was not performed on every inmate. The question is, how many who needed vaccination or re-vaccination were left unprotected.

Another essential is to know the age-distribution of these populations. In workhouses, a large number of the inmates are past the age at which small-pox attacks are likely. For example, only one-sixth of the general population reaches the age of sixty; but in workhouses the proportion of old persons is very much greater, and small-pox attack is much less likely at or over sixty than at earlier periods of life. Then, again, with regard to inmates at other ages, they must largely consist of persons who have often been in common lodging-houses, where general vaccination is attended to whenever small-pox appears, or in prisons where the operation may be a matter of routine in cases requiring it, whilst the vaccination of children born in workhouses is not likely to be neglected. These considerations make it necessary that we should know the actual facts regarding each institution cited as evidence against aerial convection.

In the absence of details, the argument from institutions does not appear to me to disprove the power of small-pox to spread by diffusion through the atmosphere.

Those who regard aerial convection as the best explanation of some of the facts we are discussing to-night may perhaps be accused of scientific credulity. But scientific incredulity may be far more dangerous. A man is apt to form very definite conclusions as to the means by which a given infectious disease may alone be conveyed; and in that case, though the disease may be spreading all around him by a different method, he, being hidebound by his preconceived opinions, will fail to recognise the agency at work. Having determined that aerial convection is impossible, or that hospitals are harmless, he may go right into the midst of an outbreak so caused, but his eyes and his mind will be closed to the facts that are before him.

THE PRESIDENT: Both sides of the question before us have been well and fully stated, and most of the points I had intended to bring forward have vanished one by one as I have listened to Dr. McVail, who has put them infinitely better than I could have done. There are still, however, one or two on which I should like to make a few remarks, and first as to the antecedent probability of aerial convection.

We are probably all agreed that particulate infective matter passes from a small-pox patient into the air of the room in which he lies, and can effect susceptible persons at short ranges, say within the room; and our faith in this would not be shaken by finding that sometimes in such circumstances actual infection did not follow. From the once frequent records of infection from rags, it may be inferred that the poison does not readily become inert, but can retain its virulence for long periods. Particulate matter of other kinds is carried far by air, in a direction determined by that of the air-currents for the time being, to settle, sooner or later, more thinly as the radius increases; and this without the aid of flies or other insects which may, nevertheless, be given. As regards smoke particles, metallic and other dust, and *bacillus prodigiosus*, these results are demonstrable. The suggestion is that small-pox particles under the same conditions behave in the same way, and like *bacillus prodigiosus* remain infective at the end of their transit. The infected air of the sick room must pass into the open air, although it is not—as smoke is—usually delivered at a high level in a hot current, with considerable upward initial velocity; and any particles it contains may therefore be expected to settle all the sooner on that account, especially in the absence of high winds, which seems to have coincided with experience of aerial convection.

It seems to be generally agreed that whatever the explanation may be, there is occasionally some "hospital influence" to be recognised: a graduated incidence of small-pox around hospitals, graduated much as the deposit of smoke would be. It might well happen, on the hypothesis of aerial convection, that owing to the direction of air-currents at critical times, the diffusion would be mainly in one direction, and that this might coincide with that of traffic to and from the hospital. But at Fulham, at all events, the larger incidence was in another direction. Dr. McVail has referred to the difficulties which attend any attempt to explain this incidence, and especially its

graduation, on the basis of personal contact or traffic. No one disputes the danger which must arise from such sources, if there be laxity of administration of hospitals and ambulances. The question is, whether the most liberal estimate of carelessness in those respects can explain the facts observed at Fulham and elsewhere, as to the amount and peculiar distribution of small-pox in the vicinity.

Why it should be only small-pox which is liable to exert this "hospital influence" is less clear on either view. As far as I know, nothing of the kind has been brought home to scarlet fever, even when isolated on a large scale in hospitals surrounded by dwellings. Measles, which has many points of analogy with small-pox, has not yet been concentrated in hospitals in the same way.

Possibly the infective matter of some of these diseases is readily destroyed by dryness, or by exposure to air, or becomes ineffective when diluted beyond a certain point. This, however, is not the only problem of the kind. Why is small-pox not conveyed by milk or water, as certain other infectious diseases are?

At all events, small-pox, and so far small-pox alone, has been found now and then to occur around hospitals in a characteristic way, the details of which would be *à priori* probable on the hypothesis of aerial convection, but could hardly have been predicted on any other view.

Negative experiences are valuable as grounds for the conclusion that aerial convection is, at all events, exceptional, which is not denied by those who accept Mr. Power's suggestion; but they cannot in themselves disprove it, any more than the escape from enteric fever epidemics of communities who derive their water supplies from polluted sources, or the escape of individuals during such an epidemic, disproves the reality of water-borne infection.

It is not a hospital question alone, though the facts can be better studied, on a large scale, when the possible focus is single, and infected in a greater degree and for a longer time. I believe that if circumstances permitted the same close observation of small-pox in private houses, we should find here and there examples of aerial convection far beyond the sick room. One suggestive instance occurred in my own experience, years ago. A case of small-pox was brought into a house in a crowded part of Nottingham, at a time when the town had long been free from the disease. It remained unreported for some days; and after the usual incubative period other cases occurred in houses near, which had, as far as could be ascertained, no direct

communication of any kind with that first infected. The secondary houses were in the same block—only a few yards away—but in courts approached only by passages from other streets. The wind was light, and blowing in the direction which the hypothesis of aerial convection would require.

We are greatly indebted to Dr. Buchanan for his excellent Paper. The discussion has been so full that he cannot speak at length to-night; but I will ask him to say a few words now, reserving for publication in the *Transactions* his reply on the discussion generally, including the observations which we hope to receive in writing from those members who have not been able to address us to-night.

The following contributions, including Dr. Buchanan's Reply on the whole Debate, have since been received:—

Dr. R. J. REECE: Although the President was so good as to invite me to speak at the adjourned discussion on Dr. Buchanan's Paper, I thought it best, in view of the lateness of the hour, and of the many references which had been made to my recent Liverpool Report, to avail myself of the Society's decision to receive additional contributions to the discussion in writing.

It may be well to point out that this Liverpool Report, as its title sets forth, is a report to the Local Government Board on Small-pox and Small-pox Hospitals in Liverpool, 1902-3, and not a thesis on aerial convection. Dr. Buchanan, in his Paper, and other speakers in the discussion, have carefully distinguished between the occurrence of what is conveniently termed "small-pox hospital influence," and the various explanations of this influence. In Liverpool the principal matter which I had to ascertain for the purposes of the Local Government Board was whether or not hospital influence had been manifested. The importance of ascertaining this is at once evident, on consideration of the situation as regards populous neighbourhoods of the three hospitals in which the acute cases were received, and of the magnitude of the Liverpool epidemic during the two years in question, Dr. Buchanan has given an outline of the principal facts as to this; fuller details can be found in my Report itself. It must also be remembered that, before I undertook my inquiries, it had been asserted that the use of these several hospitals in Liverpool had not been attended by exceptional prevalence of small-pox in their vicinity.

Thus Dr. Hope, in a Report to the Liverpool Corporation "On the Recent Outbreak of Small-pox," dated 31st December, 1903, had already said (p. 28):—

"Until within recent years there was an impression that a small-pox hospital, however well conducted, must necessarily be a source of infection to the district in which it is situated, owing to what is known as aerial convection, *i.e.*, conveyance of infection for prolonged distances through the atmosphere. It is important, therefore, that the experience of the Liverpool hospitals in this respect should be borne in mind, because it shows that in strictly-disciplined institutions, placed as these hospitals are, no danger arises from this source."

But although much importance naturally attached to opinions thus expressed, it did not appear from the data given by Dr. Hope in the Report above quoted, that they rested upon a thorough and systematic study of the local incidence of small-pox at different periods, and in relation to the operations of different hospitals: a work which necessarily would involve such enormous labour that the most energetic medical officer of health—even Dr. Hope himself—might reasonably be reluctant to undertake it. Dr. McVail has referred to a desire which has been expressed that "negative instances"—cases in which a hospital or hospitals have been stated on good authority to have exerted no adverse influence on populous areas in their vicinity—should be investigated by the Medical Department of the Local Government Board. Liverpool afforded excellent opportunities for an investigation of the kind desired. As I said in my Report (p. 10):—

"A demonstration, therefore, of absence of spread of small-pox from hospital, in the case of Liverpool, where three hospitals, two of them in the city, were receiving acute small-pox cases, and where in particular one of the hospitals (Park Hill), for a period of some twenty-four weeks at the height of the epidemic, received acute small-pox patients in large numbers, would, if established, be not only particularly interesting to epidemiologists, but in its administrative aspects would be reassuring to small-pox hospital authorities."

My main work, therefore, was to get out the data; to apply all available means of checking and correcting the collected facts as to the occurrence of cases and the invasion of houses; to determine populations and numbers of dwellings in different areas; to spot maps so that they showed, fortnight by fortnight, all the newly-invaded houses in the city; and, subsequently, to combine these various data as to local incidence of small-pox in relation to the hospital operations. In my Report I summarise the questions with which I had to deal as follows:—

"(1) Has the inhabited area within a mile in each instance of a hospital suffered more severely than the rest of Liverpool? And if so—

"(2) Has exceptional incidence within that area corresponded in point of

time (having regard, of course, to the period of incubation of small-pox) to the use of the hospital for the treatment of acute small-pox cases? And—

"(3) Is there evidence that within the several "one-mile areas," as they may be termed, dwellings nearer to hospital sustained a heavier incidence of small-pox than those further away?"

And my conclusions, based solely upon data upon the accuracy of which Dr. Hope and I are completely in accord —for obtaining many of them I am of course indebted to him and to his staff—were:—

"(1) Inhabited areas within a mile of each of the three Liverpool small-pox hospitals have suffered more severely from small-pox than the city as a whole.

"(2) Exceptional incidence of small-pox within these areas has corresponded in point of time with the use of these hospitals for the treatment of acute small-pox cases.

"(3) Broadly speaking, within these hospital areas the dwellings nearer to hospital have sustained a far heavier incidence of small-pox than those further away."

Dr. Buchanan, in his Paper, has referred to the way in which the Liverpool experience, summarised in the above conclusions, is parallel to that met with in London before 1886; and subsequently in a plurality of other instances of provincial hospitals which have had considerable populations in their neighbourhood; and with regard to Liverpool he draws the inference which I have done, namely, that we have there a notable and striking example of the characteristic "small-pox hospital influence." It is now necessary, however, to consider Dr. Hope's view of the facts brought out by my inquiries. I understand him to urge that these are not instances of true "hospital influence," but of some kind of spurious imitation of the real article; that the excessive incidence of small-pox round these hospitals, the correspondence of such incidence in point of time with hospital operations, and the graduations observed have had no relation other than fortuitous to the hospitals themselves: that, indeed, he advances a kind of "Theory of Fortuity."

I do not think Dr. Hope has assisted this contention materially by instances such as—in one series of cases —a woman, "Annie Robinson, living at No. 14, Balkan Street (which is within fifty yards of Park Hill Hospital walls)," where the source of infection was traced by his staff; for, as he himself points out, "there were no cases of small-pox in Park Hill Hospital at this time." Or in another case, which he has placed on record elsewhere, of a patient who died suddenly within the ¾-1 mile zone of the Priory Road Hospital, and to whom he traces the infection of certain other persons, for he fails to trace the infection of this primary case, and the hospital

was at the time receiving acute small-pox patients. Neither do I consider that anything is gained by the cases he quotes of Messrs. G. W. and Co., or the servant-girl at Everton Brow. Of course, scores of instances could be brought forward in which persons living near a small-pox hospital were in all probability infected independently of it. Living close by a small-pox hospital does not confer immunity from small-pox attack by direct infection—the point is too elementary to need stating. The question at issue is not whether *all* cases living within a mile of a hospital contracted small-pox from the hospital, but whether the *excessive* incidence round the hospital is attributable to the hospital.

I may add one other point. Dr. Hope succeeded—through the efforts of his staff and with the aid of the special powers to which he has referred—in tracing a probable source of infection in about 1,000 out of the 2,000 cases which occurred in Liverpool during 1902-3. Incidentally, I may note that there seems to be an ample margin for cases due to aerial convection from the hospitals. But there is also some question with regard to the 1,000 cases which are accounted for. In some of these cases, occurring in the hospital areas, it appears to me, after study of detailed lists, that after all the recorded evidence of contact with small-pox cases was slight; and that it might at least be equally probable in certain cases that aerial convection from the hospital was the real cause. This, however, can only be a matter for speculation. A further point is the possibility that certain of the undetected cases which remained at their houses (in any part of the city) caused infection in their neighbourhood as a result of aerial convection, not of direct or mediate infection. Dr. Hope has given an interesting case in point in a diagram attached to his 1903 Report on the Liverpool outbreak: where a woman named Powell, who, while suffering from small-pox, remained at a house in Lansdowne Street for three weeks before removal to hospital and is believed to have caused the infection of as many as 29 cases in the neighbourhood. Now, in accounting for these 29 cases, Dr. Hope and his staff have been content in several instances with recorded explanations, such as that the patient lived: "in the neighbourhood of Lansdowne Street," "close proximity to Lansdowne Street;" "a shop in Lansdowne Street," and so on.

This method of accounting for the infection of cases can hardly be regarded as complete. It might, perhaps, be

extended in the following manner: "Walked down a street in which the Powells lived;" "Belonged to the same Band of Hope as the Powells," "Received a telegram from the Powells."

I do not, however, seriously question that these patients did, in some way or other, receive infection from the woman Powell. But, in view of our experience of small-pox hospitals, and of cases such as that at Nottingham, to which the President has referred, it seems to me that aerial convection of infection over comparatively short distances from the Powells' house should be included among the conditions, such as *fomites* or mediate infection, which in this instance may be supposed to have favoured the local spread of the disease.

I find it somewhat difficult to follow Dr. Hope's criticism in regard to rates. The statistical part of my Report was intended for readers who could distinguish between the actual figures given and the rates calculated on those figures, and throughout that Report I have given the actual figures side by side with the rates. In dealing with the case of Fazakerley Hospital, I have in three separate places drawn attention to the necessity of caution in drawing inferences from rates based upon small numbers. The fact is, of course, that some of the rates are calculated on large figures, *e.g.*, the rates for the larger areas round Park Hill Hospital during the period 7th December, 1902, to 20th June, 1903, when that hospital was receiving cases on a large scale at the height of an epidemic, while others are calculated on small figures, *e.g.*, the fortnight-by-fortnight rates, or the rates on the small area within ¼ mile of Park Hill, which contains only 171 dwellings, nine of which were attacked, giving the rate of 526 per 10,000 referred to by Dr. Hope. The smallness of the number of houses in such an area as this, or as in the Fazakerley neighbourhood, is a matter which the epidemiologist must take as he finds. The point which comes out in dealing with the mile areas round these several hospitals, and the subdivision of those areas, is that whether you take the large figures for the large hospital at the height of the epidemic, or the smaller figures for the smaller hospital in the early portion of the epidemic, or the figures for the outside hospital with its comparatively few houses round it, each in its degree, and on the invasion rates, indicates the same characteristic excessive incidence corresponding in point of time to the hospital operations; and when the figures are largest, and the conclusions to be drawn from the rates are consequently the most definite,

the indication of the excessive incidence, and the graduation of incidence, are clearly manifest.

With regard to Dr. Hope's observations on the danger which he represents me to have attributed to Fazakerley Hospital, as compared with small-pox hospitals situated within the city, it is hardly necessary to point out that a house invasion rate of 3 per cent. in the case of a hospital like Fazakerley, which almost fulfils the Local Government Board requirements as to population, is a trifling matter administratively, by contrast with the same invasion rate in populous areas such as those round Priory Road or Park Hill.

The Netherfield Road story does not seem to me to lend much weight to the argument that the occurrences round hospitals were merely fortuitous. The analogy is hopelessly incomplete. If it is desired to show by analogy that the occurrences round the small-pox hospitals were accidents, and nothing more, we want something of this kind—a series of facts as to the incidence of small-pox round three establishments which are the only three of their kind in or near the city—say, three skin hospitals, three piano factories, or three of anything else you please, provided they are the only three, and are placed in different parts of Liverpool, and outside the small-pox hospital areas. If the analogy is to be of value, these facts must be found to show an excessive incidence of small-pox round each establishment, and a graduation of incidence round each establishment. And it would not be enough to find that this excessive incidence and this graduation occurred when the whole epidemic period was taken. In the case of establishment A, they must be found during the period in which Park Hill Hospital was receiving cases, and not at other times; in the case of establishment B, solely while Priory Road was receiving cases; and in the case of establishment C, solely when Fazakerley was receiving cases. In the absence of some such demonstration, I do not see how to attach weight to the comparison which Dr. Hope invites us to make.

What Dr. Hope appears to advocate, and what I have termed a theory of fortuity, is an explanation that the excessive and characteristic incidence of small-pox within the hospital areas was due to accident; that undetected cases, or other causes of spread of small-pox from person to person, occurred in exceptional numbers, as a result of mere chance, in the areas in question at the time when the hospitals were open. On this explanation, the apparent

relationship between the excessive incidence of small-pox on the hospital areas and the activity of the hospitals, the gradation in intensity of incidence within the hospital areas and the rest, are mere matters of coincidence.

The theory might be seriously entertained if the experience of Liverpool in 1902-3 stood by itself. But when the notorious occurrence of similar phenomena round other hospitals in other epidemics is borne in mind, it may be dismissed as altogether inadequate.

As to the explanation, my own belief is, that aerial convection probably operated in the case of each hospital. Naturally, I have carefully considered the alternative explanation: that of direct and mediate infection due to hospital communications and traffic. The data placed at my disposal by Dr. Hope give no suggestion of support to the latter explanation, in the case of any one of those hospitals. The assumptions required to explain the excessive incidence, and the graduation of incidence in each instance, by a prolonged series of unsuspected communications between the hospital and its neighbourhood, undetected by Dr. Hope's staff, seems to me to be so improbable that it may safely be dismissed. I realise, however, that in this contention I am not likely to receive the support of Dr. Newsholme.

Looking to the whole case, therefore, I remain of opinion that in Liverpool, during 1902-3, we had to deal with a remarkable series of instances of true hospital influence; the excess of small-pox in the neighbourhood of the hospitals standing to the operations of these hospitals, as Dr. Buchanan has put it, in the relation of effect and cause.

Dr. Philip Boobbyer: Owing to the interest I feel in the subject of Dr. Buchanan's Paper, and my inability to attend either of the recent meetings of the Epidemiological Society at which it was discussed, I am specially glad to avail myself of the opportunity now kindly offered me of sending in writing a contribution to the Society's *Transactions* under this heading.

At the outset I wish to say that I entirely agree with the President and other speakers, that there can be no doubt of the capacity of small-pox infection—as of other particulate virus—to spread for short distances through the air. And, given the possibility of such extension without intermediate vehicle, it is obviously difficult to limit the distance over which it can possibly take place. It is surely

a mistake, however, to consider that a virus of such exceptional—if short-lived—activity should spread almost exclusively through one only of the available media. We hear comparatively little nowadays of indirect agencies of infection, but I have known the disease undoubtedly carried on many occasions by such agencies. In the neighbourhoods immediately and remotely surrounding small-pox hospitals we have the hospital ambulances and other conveyances, and the clothing and persons of hospital servants, nurses, and medical men, continually moving about, and constituting a serious menace to unprotected persons with whom they may come into contact or proximity. This is no fancy picture of remote possibilities, but a statement of fact based upon experience. I have known small-pox carried by a coat sent for repair, after having been worn by the owner in paying one casual visit to a small-pox hospital ward, in which also he was careful to avoid actual contact with the patients and furniture. The difficulty of tracing infective contact is well known to all who have had much personal experience of small-pox outbreaks; it is also a matter of common knowledge with such persons, that the more careful and detailed the investigation the greater the proportion of cases in which such contact can be clearly traced.

Where special inspectors are employed to deal with small-pox contacts, it is often found that sources of infection are ultimately brought to light which have been missed and undreamed-of at the first inquiry, although perhaps such inquiry may have been most carefully and systematically conducted by an experienced medical man. The inspectors frequently gain the entire confidence of the people among whom they work, and thus obtain clues which might otherwise escape the most vigilant search. No one, I think, will call in question the *bona fides* or scientific expertness of Local Government Board inspectors like Dr. Buchanan and Dr. Reece, or some other recent aerialists; but all Medical Officers of Health who have had much experience in the investigation of local small-pox outbreaks, will, I think, agree with me that many important facts must almost necessarily be hidden from these gentlemen which would, in all probability, come readily to the knowledge of the local sanitary inspectors. Even two medical experts agreed upon the main issues like Drs. Buchanan and Thresh, making almost simultaneous investigations in the Essex-shore outbreak of 1901-1902, arrived at different conclusions, so far as their estimate of the possibilities of personal contact was concerned. In a recent case, too, tried

in the High Court, one medical witness adduced as evidence of air-carried infection certain cases of small-pox which were shown by another to have arisen by contact with previously known cases.

It is, I think, extremely probable that much of the infection in the Essex-shore outbreak of 1901 and 1902 was carried out of East London by dock and other labourers, travelling by road, rail, and river, to and from their work. I may instance an occurrence in my own neighbourhood, which illustrates how readily infection may thus be spread without necessarily attracting attention in the process. At the end of March, 1904, an outbreak of small-pox occurred at Arley, near Nuneaton, in Warwickshire, among some pit-sinkers there. The disease was not recognised at Arley, and several of the patients whose homes were in the neighbourhood of Nottingham returned there in a highly infectious condition to be nursed. We were fortunate enough to discover and secure the isolation of these patients; but, had we not done so, an extensive further outbreak might have occurred, and in the absence of information about the Arley cases, the source of infection in the Nottingham district would have been open to various explanations.

In looking over the zone maps, we find that on the prevailing-light-wind theory of spread, such wind must have been of very variable direction in most recorded cases. In two of Dr. Buchanan's instances, an east and a west wind are respectively necessary to explain the special incidence observed on this hypothesis. Much has been said on the subject of so-called negative evidence, with special reference to the question of its significance, but no agreement seems to have been arrived at. Now, without going the length of Haygarth in his discussion with Waterhouse, and saying that negative is practically destructive of affirmative evidence, we must all agree that it serves *pro rata* as a diluent of it; and if no evidence of aerial convection could be found by the Local Government Board in the case of four-fifths of the small-pox hospitals in use in this country; and if, as was certainly the case, the general circumstances of many of these were apparently identical with those having a positive record, then surely we are justified by such facts in asking whether it is not probably some factor more variable than the atmosphere surrounding the hospitals which is productive of the difference.

A distinguished judge to whom the problem was incidentally submitted, in a recent case tried in the High Court, insisted upon taking a broader issue, ignoring the

atmospheric question. Looking at the matter logically, he said:—"The conclusion that all hospitals are sources of danger does not necessarily follow from the premise that some are such"—with which proposition we must all agree—and then went on to add that, apart from the logical question here involved, so-called negative evidence was available to more than outweigh the positive statements on the other side. So much for a layman's view of the case.

While speaking of negative evidence, I cannot forbear giving a recent example of it from my own experience. During the past two years, it has been my practice to nurse all severe cases of small-pox occurring in Nottingham, in the open air, or rather, in bell-tents with open sides. About twenty of these cases have been so treated at our Bulwell Forest Hospital during this time. The patients have been placed within some 85 ft. of a road along which about 1000 coal-and-iron workers, and other persons, pass daily on their way to and from their work. The direction of the prevailing wind (south-west) is from the tents across the road at an angle of about 45 deg. One only of these 1000 habitual road-passengers contracted the disease, and he apparently became infected by contact with a case near his own house, at a distance from the hospital.

The actual *modus operandi* of hospital influence in propagating small-pox—when it occurs—is often stated to be immaterial; so far, at least, as administrative interests are concerned. This, however, is obviously incorrect, for if the risk of aerial spread is real and serious, then small-pox hospitals must be remotely isolated; whereas, if the principal risk is from lax administration and the like, all that is necessary for reasonable safety is to secure a site in a sparsely-inhabited district, and look carefully to the subsequent management of the institution and its inmates.

DR. J. THRESH: The subject of the aerial conveyance of the infection of small-pox has so frequently been discussed, that it is practically impossible to advance any fresh arguments either for or against; but there is no doubt in my mind, that the recent inquiries made by Drs. Reece and Buchanan, with reference to the Gateshead and Liverpool small-pox hospitals, have strengthened the evidence in favour of air-spread infection. At the same time, I am also convinced that those who refuse to accept the evidence in connection with the outbreaks which occurred in London, and, more recently, in Essex, when the London small-pox hospitals were moored in the Thames, will not have their

opinions altered by the experiences of Liverpool and Gateshead. I regret exceedingly that those who seek to explain the unusual incidence of small-pox, around large hospitals receiving acute cases, rarely produce evidence showing that they have thoroughly investigated the incidence of the disease in such cases, and have found that there was no excessive prevalence near the hospitals. This is the only kind of evidence which, in my opinion, can have any weight. Statements to the effect that no excessive incidence has been noted are freely made, but rarely supported by evidence. For example, it has often been stated that the hospitals at Liverpool had no effect upon the residents around; yet, when carefully investigated by Dr. Reece, he had no difficulty in showing that they were *foci* from which the disease was spread.

My further experience in connection with this disease, and further inquiries with reference to the effect of the hospital ships, only tend to confirm the opinion that the *materies morbi* of small-pox is conveyed in some way through the air, and may be so conveyed for considerable distances. At the time I communicated my Paper on the Essex epidemic to this Society, I mentioned that I was watching with interest the effect of the West Ham Hospital, situated in Dagenham parish in the Romford Rural District, which was then being filled with cases from West Ham and the surrounding towns. I had not long to wait, as cases speedily cropped up in the village and amongst the more scattered population around; and, although little difficulty was experienced in stamping out the disease in such densely-populated localities as West and East Ham, it could not be stamped out in this rural area, until the epidemic had practically ceased in the towns sending cases to the hospital. Myself and assistants inquired into the origin of all the cases around, and found a much larger proportion which could not be attributed to contact than was the case elsewhere. I kept a spot map, and at the termination of the outbreak found that the area round the hospital had suffered far more severely than any other part of Essex, save that near the hospital ships. In Dagenham, the parish surrounding the hospital, the attack-rate was 8 per 1,000 population; in two parishes to the south-east, lying between the small-pox ships and the West Ham Hospital, it was 25 per 1,000; in the parish adjoining Dagenham (Hornchurch), it was 3.75 per 1,000; and, in the remaining parishes of the Romford District only two cases occurred. The two parishes with the largest

proportion of cases were between two fires: exposed to the virus from the ships with the wind in one direction, and to that from the land hospital with the wind in another. The outbreak in Dagenham and Hornchurch, the parishes surrounding the West Ham Hospital, commenced when some forty to fifty cases had been received in the hospital, and continued for a month after the rapid subsidence of the epidemic in West Ham. Altogether, 123 cases occurred in the Romford Rural District, distributed as under:—

In Rainham and Warmington, between the London and West Ham Hospitals	48 cases = 25 per 1,000 population.
In Dagenham (where the West Ham Hospital is situated)... ...	49 „ = 8. „ „
In Hornchurch (the next parish) ...	24 „ = 3.75 „ „
In Havering, Upminster, Canham, and Corbetley	2 „ = .8 „ „

I could find nothing which could possibly explain this distribution, save the position of the small-pox hospitals.

Dr. G. Reid: That a small-pox hospital situated in a populous locality is, to some extent, a source of danger to the inhabitants is generally admitted, although all are not agreed as to the cause of the danger or its extent. In dealing with this question in his Paper, Dr. Buchanan fails to convey the impression that he has approached the subject with an unbiassed mind; in fact, he passes over in a few sentences the opinions of those who do not accept the theory of aerial convection as being proved. Indeed, in reading his Paper (I was not present at the meeting at which it was read), one can hardly avoid the impression that bias causes him to treat the opinions of those who differ from him rather with ridicule. The following quotation, having reference to the evidence which, according to Dr. Buchanan, some observers require in proof of the theory, I think indicates that this is so:— " . . . they appear to claim that aerial convection, if it occurs to all, must, during an epidemic, affect every house and every institution near the hospital. They are not content with a house-visitation rate of 5 per cent.; they demand that it should be 100 per cent. Having made that demand, they usually point to the escape of the 95 per cent. as evidence that aerial convection has not being operative."

While not accepting the aerial convection theory as being proved, my opinion is not based upon any such ridiculous proposition, but is the outcome of my own ex-

perience, and the experience of many others whose views are entirely disregarded in the Paper in question. Dr. Buchanan admits that there may have been examples of towns suffering from small-pox during 1900-04, where the facts proved to be negative as regards "hospital influence;" but, at the same time, he expresses his regret that he was unable to find them. It would be interesting to know what steps he took to discover such cases. Can it be possible that he had not heard of the Nottingham Small-pox Hospital action, which occupied the attention of a High Court Judge for five or six days, and in which numerous experts gave evidence both for and against the aerial convection theory, based upon previous as well as recent experience? Further, among the experts on that occasion whose evidence negatived the theory was Dr. Hope: who, in support of his opinion, instanced his recent experience in Liverpool during the same epidemic as that upon which Dr. Reece formed a diametrically opposite opinion regarding "hospital influence" and the aerial spread of the disease. Why should Dr. Reece's report receive full recognition and acceptance, and Dr. Hope's opinions be entirely disregarded, in Dr. Buchanan's Paper?

The subject is admittedly a complex one; expert opinion at the present time is probably equally divided regarding it; and the solution of the question will not be arrived at by discarding all arguments which do not fit in with one's preconceived theory, whatever it may be.

Personally, having regard to the nature of the contagion, I should have no difficulty in accepting the aerial convection theory, were it not that my own experience does not substantiate it; and when I find that the opinions of others who have had quite exceptional opportunities of judging as to the truth of the theory agree with mine, naturally I feel that my opinion is strengthened. The position as it presents itself to me is, that, while one must accept the fact that there are instances of "hospital influence," they probably can all be accounted for otherwise than by the aerial convection theory. On the other hand, there are many instances on record in which, while the circumstances have been peculiarly favourable to extension by aerial convection, no such extension has, as a fact, taken place; therefore, one cannot but question the accuracy of the theory. To my mind it is difficult, to say the least of it, to accept the theory in the face of often repeated negative experience in the case of hospitals situated in populous centres, surrounded by imperfectly-vaccinated communities;

and where, for many months at a time, and under all conditions of weather, acute cases of small-pox in considerable numbers have been under treatment.

I differ from Dr. Buchanan, and some others, regarding the comparative value of positive and negative evidence of "hospital influence" in such circumstances, as bearing on the question of aerial convection. By negative evidence we can, at any rate, prove that under conditions said to be favourable to aerial convection, such, as a fact, has not taken place; whereas, while positive evidence of "hospital influence" is no doubt met with in some cases, there are causes other than aerial convection to account for this; and because we cannot always prove that such causes have been in operation, this does not necessarily entail the acceptance of the aerial convection theory.

Like many others who now are sceptical regarding the theory, at one time I was a believer in it; but, as I have said, experience has caused me to doubt its accuracy. Without going minutely into detail, I would mention shortly what that experience has been.

We have seventeen small-pox hospitals in Staffordshire, with a total of 309 beds, serving a population of about 1,000,000; and I have records of fifty-seven distinct occasions on which the hospitals have been in use, the total number of cases isolated being 1,225.

Most of these hospitals are by no means well adapted for their purpose; and, from the point of view of aerial convection, they may be said, as a rule, to be dangerously situated. Only one complies with the Local Government Board's requirements as to distance from populations.

The following are the special features of the hospitals in this respect:—

In four cases the hospitals actually adjoin scarlet-fever hospitals; in two cases the hospitals are within 100 and 150 yards respectively of workhouses; in eight cases the hospitals are within 100 to 300 yards of populous centres; in eight cases the hospitals may be said to be actually surrounded by groups of houses.

Notwithstanding the above facts, I can confidently state that on none of the fifty-seven distinct occasions on which these hospitals were in use has a single case of small-pox occurred which, by any possibility, could be attributed to aerial convection from the hospitals. In one instance (the only one which occurred) an attack of small-pox was traced to one of the hospitals; but the patient, who was a road man, had been working for a week or two on the road

adjoining the hospital; and it was conclusively proved that he had frequent conversations with the patients, and had been in direct contact with some of them. This occurred at a time when the hospital was greatly overcrowded and understaffed, and the single protecting fence did not constitute a sufficient barrier against communication with the hospital from the outside.

I may mention that from this case others arose before the disease was recognised; and, had the origin of the first case not been traced to direct contact, aerial convection would no doubt have been credited with the occurence.

Possibly it may be suggested that some of the hospitals referred to may have had too small a number of beds to justify much weight being attached to the absence of evidence of aerial convection; and, as I have no desire to attach more weight to my argument than the facts warrant, I have grouped the hospitals as follows, to show the actual number of beds. It must be remembered, however, that overcrowding frequently happened, and larger numbers of cases were admitted than the number of beds warranted: temporary provision being made for their accommodation:—

No. of Beds.	No. of Hospitals.
5 to 10	7
12 to 16	4
17 to 22	4
60	2
	17

DR. R. D. SWEETING: Whilst generally agreeing with Dr. Buchanan in his conclusions, I think that there is danger of overstating the case for aerial convection, by assuming that graduated intensity of infection around a small-pox hospital *per se* necessarily connotes aerial convection. In this connection, I cannot do better than reproduce the conclusion of the Royal Hospitals Commissioners of 1882 on this point. They say, on p. 26 of that Report:—

"One point appears to have made so much impression on several important witnesses that it should be specially noticed. Mr. Power himself clearly perceives, and fully admits, that the graduated distribution of disease around the hospitals, which is so remarkably illustrated in his report, is as explicable by personal communication as by atmospheric dissemination. *With such a hypothesis*, he says—meaning that of dissemination—*equally as with a hypothesis of conveyance by human movements, the gradation of hospital influence from centre to periphery would be in complete accordance.* The admission is so material that it ought to be developed. Let us replace the image of a wave, which is suggestive of one hypothesis, by an image equally appropriate, but

adjusted to the other. Instead of an expanding wave, let us imagine a number of converging and diverging lines of infection. It is plain, in the first place, that along these lines—the lines of hospital communication—the incoming and outgoing of persons and things would offer more occasion of disease wherever these persons or things were likely to pause in their route, and by consequence more in the immediate neighbourhood of the hospital than farther off. But disregarding this effect of propinquity, and supposing that one quarter of a mile is as fruitful in occasions of contact with the carrier of infection as another, let us imagine that, on the different lines of communication which radiate to and from the hospital, infection is shed equably along the whole of their courses, and is represented on a diagram by black lines, varying in breadth according to the amount of infection which is dispersed, and in length according to the distance along which it is carried. It does not need the actual inspection of such a diagram to perceive that the effect of these lines in blackening each successive hospital ring will constantly increase as they close in on the centre; or, in other words, that the converging incomings and outgoings of the hospital would produce exactly the 'graduated intensity of infection,' from which, prior to reflection, we are tempted to infer an expanding wave. The argument is capable of arithmetical statement, and it would be easy, if it were worth while, so to exhibit it."

It is, therefore, clear to me, following this able and lucid statement of the Commissioners, that all that graduated intensity from centre to periphery of the mile circle around a small-pox hospital shows is *hospital influence.* But this may be aerial or administrative; and, therefore, it comes to this: that every instance of alleged spread from a small-pox hospital must be taken on its merits and fully invesvestigated, before it can be said that aerial convection is fully established. Mr. Power did this in his 1884 Fulham Inquiry, when he conclusively eliminated administration. Dr. Buchanan has done this also to a large extent in regard to Gateshead and Purfleet; whilst in the Liverpool case of Dr. Reece, though administration is not formally excluded in the Report, this mode of spread was expressly eliminated by Dr. Hope, the Medical Officer of Health, whose position in this respect was taken as a postulate at the outset of the investigation. In brief, where you have graduated incidence from centre to circumference around a small-pox hospital, you must, by inquiry and reasoning, eliminate administration before you can prove—or even suggest with any force—aerial transmission. The graduated intensity of itself does not prove conveyance of small-pox infection through the air.

I cannot accept the view that the immunity of the district around Clayton Vale Hospital was due to the carbolic oiling of patients: for this reason that, at Fulham in the 1880-1881 epidemic, this was carried out to a large extent as matter of routine; whilst in 1884-1885, at the same hospital, when the numbers were expressly limited to a dozen or so, the oiling process was carried out

very fully and thoroughly. Masks, too, were often used, *e.g.*, of various substances, intended to prevent pitting. I cannot but think that the non-spread of small-pox from this hospital was rather due to non-aggregation of acute cases and paucity of susceptible material around the hospital. At any rate, it is a negative instance only, and of little value one way or another.

Finally, my experience at Fulham in 1884-85, when small-pox and fever were treated at one and the same time, on opposite sides of the hospital, and separately administered, is opposed to Dr. Ker's at Edinburgh. Cases of small-pox repeatedly cropped up in the scarlet-fever wards amongst imperfectly-vaccinated children; and it was only by re-vaccinating promptly all scarlatinal cases directly their temperatures dropped, that control was enabled to be exercised over these manifestations. Here, again, though aerial convection was highly probable, administration could not be altogether eliminated; for, in spite of elaborate precautions, I was accused of introducing small-pox into the fever wards, being the only person who visited both parts of the hospital.

PROFESSOR KENWOOD: The impression left upon my mind after the perusal of Dr. Buchanan's Paper was one of appreciation of the able manner in which he had handled certain facts, in an endeavour to make them prove his case for the distal aerial convection of small-pox. He even went to the extent of using the negative instance of hospital influence at Manchester as an argument in favour of this theory, without stopping to consider whether the precautions adopted in that hospital did not also operate against the dissemination of the disease by human agency. All must acknowledge that, in some recorded instances, a small-pox hospital has been the centre of a zone of exceptional incidence, and that this incidence has in some cases been graduated. The real point at issue is as to how the graduated zone of incidence, which is *sometimes* observed, is best accounted for. In the opinion of many of us, the manifold channels of direct and mediate infection through human agency offer the most acceptable explanation of all the hitherto-recorded facts.

In seeking for the true explanation of the nature and extent of hospital influence, it is necessary to keep in mind the facts that it frequently does not exist at all; that small-pox epidemics are generally characterised by the establishment of series of zones with graduated incidence, quite

irrespective of the locality of small-pox hospitals; and that the zone near the hospital is generally inhabited by the poorer section of the community. Small-pox hospitals are not placed in good-class residential districts; and the conditions of life, of housing, and of vaccination, among the poorer people, lend themselves to an increased incidence of the disease. It is the experience everywhere that the poorer part of the community suffers from small-pox generally two or three times more than the better-class part; therefore, is it fair to compare the incidence upon a broad hospital zone with a one-mile radius (and, therefore, over three square miles in area) with that of the rest of the town, which includes the better-class residences and residents?

Although there is a tendency to exaggerate the true extent of hospital influence, I have no doubt that it has been in existence in some cases. But the fact remains that for every instance of alleged hospital influence it is easy to produce at least one instance (I believe I could produce two) where no such influences existed. That circumstance seems to me to tell most strongly against the theory of distal aerial convection. The aerial convectionists appreciate this difficulty, and they meet it by surmising that the favourable conditions for aerial convection are rarely in operation. Well, from the scant meteorological information to be gleaned in different epidemics, I have observed that at the time when aerial infection is claimed to be in operation, the most diverse atmospheric conditions have prevailed, such as high winds, low winds and calm; clear, blue sky and fog; keen frost and high mean temperature. Dr. Savill has shown that at Warrington the incidence of the disease was not greatest in the direction to which the prevailing winds blew; and Dr. Newsholme, in criticising the Liverpool experience, points out that the prevailing winds must have been blowing in opposite directions at the same time, if they were instrumental in carrying small-pox infection to two of the hospitals which are alleged to have shown hospital influence. It is to my mind preposterous to tell us that the infection can be carried for a mile, but that the necessary atmospheric conditions in our ever-changing climate may be unfavourable for the period of nine months after acute cases are admitted to the hospital (as at Liverpool); and that it may not come into operation at all throughout the whole epidemic, in at least as many instances as it does do so, even where (to adopt Dr. Buchanan's favourite formula) "there is no evidence" of any difference as to

vaccination among those who occupy the mile-zone and those who live beyond it. Surely, if the theory is true, the operation of cause and effect should be observable in every case where a fair opportunity is presented for the experiment. Dr. Buchanan's travesty of our position in the matter has been referred to by Dr. Newsholme. Nobody in his senses maintains that every individual within the mile-zone should catch small-pox; but we do say that *some* of the susceptible people should do so in *every* instance where the hospital is occupied by many acute cases for many months. This is what I understood Mr. Justice Farwell argued in the Nottingham case; and, if so, Dr. McVail's criticism is unjustified, because it is based on the assumption that the learned Judge's argument that a negative instance unexplained spoilt the chain, had reference to infection from an individual, instead of to a negative instance of distal aerial convection from a small-pox hospital.

The operations of human intercourse are often untraceable, and they necessarily vary with the efficiency of the hospital administration. It is never practicable to exclude all the possibilities of mediate infection due to human intercourse; and those of us who have had much experience of small-pox appreciate how difficult it is to define the set of conditions which may determine the spread of infection; and we have had many opportunities of noting how the element of chance appears to come in: so that on two occasions, which appear to be comparable in every respect, the infection gets a hold in one case and fails to do so in the other. Some of the arguments advanced appear to suggest that because you cannot trace the human agency (direct or mediate) in the spread of infection, aerial spread is the only possible explanation. Well, during my thirteen years' experience as a health officer, I have not succeeded in tracing the channel of infection in quite one-fourth of the cases of scarlet fever, diphtheria, and other communicable diseases that I have investigated. Am I to console myself for my failure by such a theory as that of distal aerial convection? Faulty administration, the wilful withholding of information, accidental contact with unrecognised (and, therefore, never known) cases, imported infection, and the many circumstances favouring spread which obtain most among the poorer classes, all combine to account for small-pox hospital influence. When I hear anyone denying the possibility of leakages and of breaches of hospital regulations, I envy him his faith in things human;

and I think of the simple faith that once was mine, and which has been so rudely shaken by my subsequent experiences of hospital administration.

Dr. Buchanan asserts that there is "no evidence" that personal communication between the hospital and the quarter of a mile zone is greater than that between the hospital and the more distant zone. It is difficult to believe that he states this seriously. The hospital is the centre, during an epidemic of a large amount of traffic (ambulances, visitors, staff, workmen, tradesmen, etc.), which converges, and therefore becomes more and more intensified in each successive zone as the hospital is approached. I see no difficulty in accepting this circumstance as the explanation of the graduated incidence which is sometimes observed; especially when it is borne in mind that the communication with the hospital staff is always greatest nearest the hospital. Despite what Dr. Buchanan asserts, we generally, in my experience, draw some of our staff from very near the hospital; and, in respect to this point, Dr. Goodall has forwarded me the following statement:—

"I think it is somewhat strong to make a general statement that hospital staffs are not recruited from the neighbourhood of the hospitals. I am sure that this cannot be said of the fever hospitals of London, except with respect to the nursing staff. At Homerton, at any rate, a considerable number of the ward-maids, laundry-maids, porters, etc., come from the immediate vicinity of the hospital. Even if they have not relatives, they have friends quite close to it. And these classes of staff all come in close contact with infection in the hospital.

"Of recent years the Asylums Board, in order not to maintain a too numerous staff, have systematically transferred members of the staff from one hospital to another. I have had many such at Homerton from other hospitals of the Board. Now, it is quite common for these people to resign after being with us for a few weeks; and a frequent reason given for resignation is: 'This hospital is too far from my home; and I want to go back, if possible, to —— Hospital, because my friends live near it.'

"In epidemic times a staff usually has to be got together in a hurry; and it would be extremely inconvenient, if not difficult, with respect to these lower grades of the staff, to make sure that applicants from the immediate vicinity of the hospital were excluded."

Before the theory of aerial convection is accepted there should be unequivocal evidence of its soundness, because of the momentous issues which are involved in its acceptance. It should be demonstrated that the usual methods of spread of infection can be excluded (if that is possible); that the number of homes in which the infection cannot be traced to the usual channels diminishes with the distance from the hospital; and that the localised outbursts within the hospital mile-zone are *always* to be observed when there are many susceptible persons living for several months near

a hospital containing many acute cases. It is not permissable to lump together all the infected houses, whether they have been infected by human intercourse or not, and claim that the results prove aerial convection, when they at most only indicate the origin and spread of the infection. How such a practice may mislead has been alluded to by Dr. Hope; and Dr. Clayton (the Medical Officer of Health of Gateshead) claims that of the 56 cases within half-a-mile of the small-pox hospital, the infection was traced to human agency in all but four cases.

Dr. Reece's excellent slide demonstration showed us a long latent period of some nine months, during which patients were admitted to the Priory Road Hospital at Liverpool before any marked incidence occurred in the neighbourhood. Prior to this, there was a localised outbreak in a crowded area in the heart of the city, and from this centre the infection spread towards the hospital zone; and there, among "a slum population," it lingered and persisted. Moreover, the increased incidence in the hospital zone was synchronous with excessive incidence in the other hospital areas, *and in Liverpool outside the hospital areas*; and the incidence of small-pox upon the Parkhill Hospital area had set in shortly before the hospital commenced to receive patients. This does not appear to me to be very convincing evidence even of hospital influence, much less of aerial convection.

An argument adduced by Dr. Niven against human agency being the explanation of the mode of conveyance of infection from a small-pox hospital is, that he regards the infection of contacts as unusual and difficult. My experience on the whole supports this, although I have known several instances which point to the opposite conclusion; but the patient, before he gets into the hospital, has often the acute stage well developed; and the circumstance that so many of those who come in contact with him escape infection, though not protected by vaccination or re-vaccination, appears to me to tell against the theory of distal aerial convection. If the striking power of the infection is limited so near its origin, is it likely to be operative when more diluted and at the distance of a mile?

Hutchison's experiments with *B. prodigiosus* sprayed into the air of the parade ground at Göttingen, and recovered nearly 2,000 ft. away, is a remarkable one, but I do not see that it is any argument in favour of the distal aerial convection of small-pox. Assuming the observation to have been made with all the necessary control experiments (for

prodigiosus is commonly found in air), how long did it take to cover the distance of one-third of a mile? Under an hour, I believe: *not nine months!*

It is contended that because you do not get hospital influence in scarlet fever, and you do in small-pox, there must be some material difference in the quality of the infection of the two diseases. The contention is reasonable, but it tells no more in favour of distal aerial convection than of the alternative view.

A most interesting discussion has resulted in some new light being shed, and some fresh arguments adduced, on both sides. It is sincerely to be hoped that when further opportunities of testing this matter occur, the fullest advantage will be taken of them, and that the inquiry will be conducted in a scientific spirit and devoid of all bias. Meanwhile, I contend that the theory of distal aerial convection has never been justified by the data which have hitherto been adduced in support of it; and that is the reason why it does not appeal to the general body of Medical Officers of Health or to His Majesty's Judges.

DR. WELLESLEY HARRIS: I listened with considerable interest to the discussion raised by Dr. Buchanan's Paper on this important subject; but could not find anything in the statements of the speakers who so forcibly ridiculed its possibilities to, in any way, explain how various extensions of the disease which had come under my observation could have occurred except by aerial convection.

The examples to which I would refer occurred during the period—nearly twelve years—I was Medical Officer for the borough and port of Southampton.

For several years the borough relied upon two private houses situated in the poorest quarter of the town, which they had converted into what they were pleased to term a Small-pox Hospital; and it was found that when four or five cases only were isolated the disease did not spread in the neighbourhood, but whenever a large number of cases were housed in the hospital at the same time, the disease quickly showed itself among the residents of the streets in the immediate vicinity.

Now it is an important fact that in the years 1891, 1892, 1893, and 1894, the first cases of small-pox in Southampton were all imported by ships, most frequently from the West Indies and the River Plate; and in those instances where cases occurred in the town they were subsequent to the isolation of a number of cases from ships in the Hospital.

The question of contact by outside people with the staff can be absolutely disregarded. Special arrangements were made for delivery of food, by which tradesmen were prevented from coming into contact with the staff. The nurses and the other staff consented to forego "leave," and refrained from entering the town while cases were in the Hospital, special arrangements being made for their recreation by means of boating, access to which was gained by a private landing-stage at the rear of the Hospital.

So strong was the opinion that the Hospital was responsible for the repeated extension to the immediate neighbourhood, that a floating hospital was provided for the isolation of small-pox cases, and the land hospital abandoned.

It is strange if it should be by mere coincidence, after the establishment of the floating hospital on the broad estuary of Southampton Water, some two miles from the town, that no further extension of small-pox to the town occurred from imported cases; and in one instance as many as sixteen cases of a severe variety were removed from one ship.

I think the arguments used by one speaker, Dr. Hope, and the spot maps entirely fallacious. To merely spot out positions in which cases of small-pox have occurred in a town, and to draw circles and say that if small-pox hospitals had existed in those districts they would have been blamed, seems to me an unreasonable argument.

I also fail to see the force of a statement which was made by one speaker that, in regard to the aerial convection of small-pox he took up the position of an agnostic, and would not believe it until it was proved. Surely, rather the reverse position should be taken, and aerial convection believed until disproved. This is the view I think the speaker would adopt with regard to other principles.

Personally, I still believe that the aggregation of a number of cases, or severe cases, of small-pox in badly-situated hospitals leads to the spread of the disease by aerial convection to persons residing in the neighbourhood. Whether this be right or wrong, I don't think there can be any doubt that it must be an undesirable practice to establish small-pox hospitals near populous districts.

If this is agreed, what does it matter whether those who believe in aerial convection are right or wrong, as long as the public are safeguarded from the dangers which follow from what, I venture to think, is an improper form of isolation As far as I am personally concerned, far weightier

evidence than that which has been offered up to the present must be produced before I can alter the opinion which is ingrained in me by the experience I have had.

LIEUTENANT-COLONEL A. M. DAVIES: There seems to be a reluctance to accept the theory of aerial convection; but, with a movement of air which is only just perceptible, a distance of one mile would be traversed in half an hour. Dr. Thresh showed that there had been a decided wind-prevalence, blowing from the Hospital Ships in the direction of Purfleet: to have been noticeable, this velocity must have been at least eight miles an hour, at which the distance (three-quarters of a mile) would have been traversed in less than six minutes. If the air of a sick-room in which is lying a small-pox patient (or 100 such patients) contains infective material—as to which, I suppose, there is no doubt—why should we conclude that the infectivity of the particles in that air is lost six minutes, or even half an hour later, merely because it has been carried a few hundred yards, or even a mile or two? With a distinct but not strong wind blowing in the given direction, the air, with its contained infected particles, would traverse the three-quarters of a mile in less than three minutes. An outbreak of enteric fever at Quetta, India, in 1898, was almost conclusively traced to air-borne infection, conveyed a distance of about 2000 yards by prevalent winds; the *contagium* of small-pox is probably more resistant than that of enteric fever to sunlight and desiccation.

The chief argument against aerial convection, to my mind, is that adduced by Dr. Newsholme in reference to the Stockwell Hospital, where a spot map showed the usual concentration of cases towards it; but only *on one side*, a railway on the other presenting no obstacle to aerial convection, but an insuperable barrier to human intercourse. In the Essex outbreak the Thames, through a barrier to human intercourse, did not impede the carriage of infection. Are there any other examples to corroborate that of Stockwell?

If Mr. Henman's device of filtering the air leaving a hospital ward, in connection with the Plenum system of ventilation, were carried out, small-pox hospitals would presumably be deprived of their dangerous characteristics, so far as aerial convection is concerned. Has this been practically tried anywhere? It would surely be worth while to make an experimental installation, which might settle the question.

DR. J. T. C. NASH: I regret that the exigencies of time have prevented me from hearing any part of the discussion on Dr. Buchanan's able Paper. The general facts adduced by Dr. Buchanan have, to my mind, materially strengthened the "aerial convection" theory so ably advanced by Mr. Power.

So eminent, however, are the authorities behind this theory, that there is a danger of its becoming blindly accepted as proven; but the true scientific spirit recognises that the greater the authority the more necessary is it to subject all the particulates to keen criticism and unbiassed judgment.

To criticise Dr. Buchanan's data in some detail:—

Surely, the excessive incidence in the very large district of Orsett should hardly be ascribed to the influence of the Hospital Ships. Yet Dr. Buchanan compares the attack rate in the twelve rural parishes comprising the very extensive rural area of the Orsett Union, which was 18.5, and that of the urban district of Grays (a riverside town no less than three miles below the ships), which was 16, with the attack-rate in London, which was only 2 per 1000. Are the districts or conditions really comparable? Dr. Buchanan and Dr. Thresh agree that the districts concerned were well organised to deal with small-pox in the way of isolation and disinfection; but how about that most important of all measures in the *prevention* of small-pox, viz., antecedent vaccination? In the vaccination returns for the county of Essex, recorded in the Thirty-First Annual Report of the Local Government Board, 1901-2, we find that nearly 50 per cent. of the children born since 1893 were unvaccinated in 1901. Anti-vaccination is notorious in this district, fostered by an anti-vaccination press. The number of re-vaccinated persons in the district must have been very small. Want of vaccination was, in my humble opinion, the chief reason why there was an exceptional incidence of small-pox in the Orsett district in 1901-2.

With respect to the cases occurring among workmen employed in erecting a temporary hospital on the flats on the Kent shore, it is admitted that personal communication between members of the staff of the hospital ships and their workmen occurred. No further comment is therefore necessary.

With respect to cases of small-pox occurring among the crews of vessels which anchored in Long Reach, surely these could be more easily explained by surreptitious visits

ashore at Purfleet, Grays, Aveley, etc., than by the proximity of the hospital ships. In my own district a case of small-pox occurred in December, 1901, which I traced to a visit to a house in Aveley, in the Orsett Union, from which two cases of small-pox had recently been removed to hospital.

In 1902, again, I had a case occurring in an unvaccinated girl, whose father, I ascertained on inquiry, had recently been working on the Kent flats, near Dartford. The father was vaccinated, but had carried home infection to his unvaccinated child. The father was a disbeliever in vaccination, and also seemed to have a contempt for common precautions. Judging by his statements, a fairly free communication took place between the workmen and small pox contacts.

Finally, it is notorious that the Metropolitan Asylums Board permitted friends to visit patients on board these Hospital Ships with a freedom not above criticism.

In view of the foregoing statements, I cannot agree with Dr. Buchanan that the incidence of small-pox in the Orsett Union, on the Kent shore, and on the crews of vessels anchored near the Hospital Ships, affords so much support to the aerial convection of small-pox as he and Dr. Thresh assume.

On the other hand, in view of the undoubtedly greater contagiousness of small-pox and chicken-pox, as contrasted with scarlet fever, I am quite prepared to admit that, occasionally, a small-pox hospital may be responsible, to some extent, for aerial convection: more particularly when aided by those notorious germ-carriers—flies.

Glancing at the tabular statement of Glasgow small-pox in Dr. Buchanan's Paper, one cannot but notice that in 1894 the largest percentage of cases came from the central administrative district, and in 1895 from the west district; so that the greatest incidence in two out of the seven years was not in the immediate proximity of the Small-pox Hospital in the east district.

Dr. Clayton, Medical Officer of Health for Gateshead, has shown* that the great majority of cases occurring there could be traced to direct contact, and, therefore, these cases fail to support the theory of aerial convection.

The Manchester evidence is directly negative.

After reviewing Dr. Reece's Report on the behaviour of small-pox in Liverpool, Dr. Buchanan speaks of "hospital

* *Journal of the Royal Sanitary Institute*, vol. xxvi, No. 4.

influence," a much safer term than aerial convection; but unfortunately, he swings back to the hypothesis of aerial convection as the explanation of hospital influence. For myself, apart from the agency of flies, I think the thesis of aerial convection is still not proven.

Insect agency, human imperfections, and errors of administration, no doubt account for some instances of undoubted "hospital influence"; and for these reasons I think the Local Government Board is wise in insisting on the isolation of a small-pox hospital.

Further, there is reason for thinking that the infective agent of small-pox is a spore-forming protozöon, and therefore capable of resisting adverse influences, such as fresh air and sunlight, to a greater extent than—for instance—the typhoid bacillus or the diphtheria bacillus, and the probably non-sporing bacterium of scarlet fever.

Therefore, one would expect more frequent instances of the aerial convection of small-pox than of the other chief infective diseases; but while I am quite prepared to admit, on the grounds I have just named, that "hospital influence" is more likely to be met with in cases of small-pox than with the other infectious diseases, I still think that the majority of cases, occurring outside a quarter-mile or half-mile radius of any small-pox hospital, arise quite independently of any hospital influence.

DR. HAMER: Dr. Buchanan has pointed out that at Felling and Purfleet "hospital communication and traffic" were practically non-existent; and that, none the less, exceptional incidence of small-pox in the hospital neighbourhood occurred. Local peculiarities render these two instances particularly instructive; but it may be observed generally, apart from special circumstances, that the fact that hospital influence continues to manifest itself with apparently unabated vigour under twentieth-century conditions, is in itself an important one. "Hospital communication and traffic" at the present time, and in the 'seventies and early 'eighties of the last century, are not one and the same thing. The mischief, if not altogether done away with, has at least been reduced within very narrow proportions by precautions such as are nowadays taken. Mr. Power, at Fulham in 1881, was able to set on one side this source of risk, as not in itself sufficient to explain the phenomena observed; and in comparison with the difficulties presented then, those met with in the modern instances must be regarded as insignificant.

Another aspect of the Purfleet case, which was referred to by Dr. Reece, is deserving of study. The "hospital communication and traffic," which can be excluded from consideration in Purfleet itself, were operative, of course, mainly upon the population of London. Indeed, an experiment on a large scale has been in effect carried out since 1885 upon this population; for in London the influence of hospital communication and traffic, exerted during the last twenty years, can be studied almost entirely uncomplicated by any influences exercised by aerial convection.

Sir Shirley Murphy's diagram, exhibited by Dr. Reece, shows that in precise and abrupt correspondence with the inauguration of the new departure of removing small-pox cases to Long Reach, small-pox ceased to prevail in London on the scale exhibited when the hospitals were within the metropolitan area.

DR. BULSTRODE: Certain of the speakers have expressed the view that the remarkable graduation in the incidence of small-pox which has been observed on so many occasions around small-pox hospitals could on general grounds be as well explained upon a thesis of personal infection as upon a thesis of aerial convection; the implication presumably being that the history of each case *quâ* exposure to infection, etc., must be carefully inquired into before any conclusion can be arrived at. Although this statement is in some degree true, it needs, I would suggest, material modification before it can be accepted in its entirety. The statement as it stands can only apply to those instances in which the topographical and social conditions around the hospital are such as to allow equal opportunities for personal infection in all directions: a circumstance which is but rarely found. A graduated intensity might perhaps be expected as the result of personal infection on the main road of ingress and egress from the hospital, but not, I think, in quadrants; wherein the streets could only be reached by a long and circuitous route from the hospital gates. It would not, I expect, be seriously contended that in the repeated outbursts around the Fulham Hospital the distribution of the cases could be, *à priori*, as well explained by personal infection as by aerial means.

Other speakers have implied their intention of not accepting aerial convection as *a working hypothesis*, basis and type, until it can be definitely proved. These are the agnostics, and doubtless the proof with which alone they will be satisfied may be long in coming. Logically,

however, these speakers are also agnostics with reference to the nebulous hypothesis, the atomic theory, and the undulatory theory of light; and to be consistent they should refuse to take note of these theories in their endeavours to explain natural phenomena, to which, in the opinion of most physicists, these theories afford a key. None of these theories have yet been, or are likely to be, proved; but those who refuse to accept them must surely find themselves in a somewhat difficult position.

The question with regard to aerial convection is not as to whether it has been proved, but as to whether, in the present state of our knowledge, it is the best working explanation of the peculiar phenomena which have been repeatedly observed around hospitals devoted to the isolation of *small-pox*, but not around hospitals devoted to the isolation of *other infectious diseases*. No one has, so far as I am aware, offered any explanation of the fact that no special incidence of infectious disease has been observed around hospitals devoted to infectious diseases other than small-pox. Surely, if the behaviour of small-pox around small-pox hospitals can be in all instances explained by personal infection, it would be reasonable to anticipate that some such manifestation would be sometimes observed around hospitals devoted to other infectious diseases.

Some of those who have been the most strenuous opponents of aerial convection would appear, at any rate until quite recently, to have regarded this theory not only as improbable, but even as largely inconceivable, and they have lost no opportunity of casting ridicule upon it. To my mind, there are numerous facts which suggest not only its possibility but its probability. Indeed, I personally see nothing contrary to expectation (to quote Paley) in the theory, either on zoological, meteorological, or statistical grounds. To ask for proof is to place oneself beyond the range of criticism. What outbreaks have been conclusively *proved* to have been caused by any definite agency? Surely, it is almost always a question of degrees of probability.

There would seem to be every reason, arguing on analogy, for assuming that the virus of small-pox is a micro-organism of more than ordinary resistance. Its high infectivity, and its admittedly long striking distance, would suggest that on these points this disease should be placed almost in a category of its own. Assuming, therefore (upon evidence which surely most epidemiologists would accept), the special resistance of this organism, what is there to prevent its carriage by the wind, when mixed

with other particulate matter, for distances with which the ordinary demands of the aerialist are but trifling.

Darwin, in the *Voyage of the "Beagle,"* refers to the falling of fine dust upon the decks of ships far out in the Atlantic; and some of the dust, which he himself collected, was found to consist, in great part, of infusoria, with siliceous shells, and of the siliceous tissue of plants.

In five little packets which Darwin sent to Professor Ehrenberg, no less than sixty-seven different organic forms were found. Darwin adds:—

"The infusoria, with the exception of two marine species, are all inhabitants of fresh water. I have found no less than fifteen accounts of dust having fallen on vessels when far out in the Atlantic. From the direction of the wind, whenever it has fallen, and from its having always fallen during those months when the Harmattan is known to raise clouds of dust high into the atmosphere, we may be sure that it all comes from Africa."

As regards the retention of the vitality of seeds when carried by one or another agency from one land to another, suggestive references will be found in the chapter on the geographical distribution of species, which is contained in Darwin's *Origin of Species.* Alfred Russell Wallace, in his *Island Life,* when writing on the dispersal of seeds, says;—

"The seeds of plants are liable to be dispersed by a greater variety of agents than any other organisms; while the tenacity of life under varying conditions of heat and cold, drought and moisture, is also exceptionally great.

"An immense number are specially adapted to be carried by the wind, through the possession of down or hairs on membranous wings or processes; while others are so minute, and produced in such profusion, that it is difficult to place a limit to the distance they might be carried by gales of wind or hurricanes."

In the *Quarterly Journal* of the Royal Meteorological Society, January, 1904, vol. xxx, No. 129, there is an account by Mr. H. R. Mill, D.Sc., and Mr. R. G. K. Lempfert, M.A., of the great dustfall of February, 1902, the evidence pointing to the conclusion that the dust which was deposited so widely over the South of England was brought by the winds from the North-west coast of Africa.

The nature of this dust was investigated by Mr. J. S. Flett, M.A., D.Sc., and it was found that—

"Plant *débris* was very abundant: Vegetable hairs occurred everywhere often in great quantity; fragments of epidermis and of cuticle, siliceous skeletons of the epidermis of grasses ..... spores of cryptogams (either singly or in masses), spore cases, resting spores of fungi, hyphæ and various unicellular or filiform, algæ, were all to be found. Diatoms were not frequent—in fact, they were comparatively rare, as were also desmids. Many of the samples sent in a moist condition were full of bacteria."

I have given these extracts, not because I wish to imply that small-pox can be carried over the long distances above referred to, but in order to furnish evidence pointing to the probability of the resistant seeds (vegetable organisms) of disease being carried with other particulate matter over, at any rate, a mile or so from hospitals.

I doubt, however, after Dr. Buchanan's admirable Paper, whether much will be heard as to the *impossibility* of aerial convection.

The following extract from the *Lancet* of July 23rd, 1904, is also of interest in relation to the carriage of soot in the direction of the prevailing wind:—

"Dr. William Butler, the Medical Officer of Health of Willesden, has made some interesting observations upon the smoke and dust nuisances in this district. Allegations were made as to the dust which was stated to come from the Metropolitan Electric Supply Works; and, with the view of throwing light upon this question, Dr. Butler procured twelve boxes, each of the superficial area of one square yard and of a depth of six inches. The bottoms of the boxes were covered with glazed American cloth, to facilitate the removal of accumulations. These boxes were placed in the back gardens at twelve different sites, and in such a position that whatever was deposited in them from the atmosphere would be retained. This dust was collected every twenty-four hours and placed in labelled pill-boxes, and upon examination it was found to consist of black carbonaceous particles distributed among a much finer black powder. These particles consisted of 'miniature cinders,' or the incomplete combustion of fine coal. Extraneous particles were, so far as practicable, separated and the coal dust was weighed, and it was found that substantial deposits were only to be found in those places which lay from the chimney in the direction of the prevailing wind, and the nearer the chimney was approached the larger was the quantity of dust collected."

There is one instance illustrating graduated incidence around a small-pox hospital which appears to have escaped notice by many of us; and it was only quite recently, while looking through some old Reports of medical officers of health, that I discovered in the Annual Report for 1899 of Dr. Wright Mason, of Hull, a diagram which illustrates the point in question. It will obviate any misunderstanding as to the purport of the diagram, if I abstract in full Dr. Mason's observations with regard to it.

Referring to the Garrison Hospital in which the small-pox cases arising in Hull were treated, he says:—

The accompanying diagram shows the percentage of houses of which one or more of the inmates were attacked by small-pox in the north-east, north-west, and south-west quarters, and in the various zones, taking the hospital as a centre. It will be observed that in the south-east quarter no cases are shown. The hospital was situated in close proximity to the river Humber, and in this quarter no houses intervened between the hospital and the river. Within the quarter-zone there were only some seven houses, and of these one was invaded. On account of the numbers being so small, it is not thought advisable to show the percentages and the zone.

It will be noticed that in each quarter the percentage of houses invaded

becomes progressiveiy less as the distance from the hospital increases, with the exception of the "three-quarter mile" zone in the south-western quarter, which district is almost entirely occupied by docks, warehouses, and railway goods-stations.

It may be pointed out that of the 305 days from March 2nd to December 31st, the Meteorological Report shows the direction of the wind to have been from the south-west on 84 days; and it will be noticed that the percentage of invaded houses was much greater in the north-east quarter than in others, this being the direction in which the south-westerly wind would blow after passing over the hospital. The next more-prevailing winds were north-westerly, which occurred on 53 days, so that the number of days on which a south-westerly wind prevailed exceeded by 31 days the next most prevalent.

Dr. CHALMERS (Glasgow) has forwarded advance pages of his forthcoming Annual Report to the Corporation of Glasgow, and he has marked two paragraphs among those which deal with the recurrence of small-pox in Glasgow during 1903-4 as a contribution to this discussion.

Interval of Freedom from the Disease.—Between August, 1902, and September, 1903, only one case of indigenous small-pox occurred in Glasgow. This occurred in May, 1903, in the wife of a seaman who himself had developed the disease a few days after his return from a voyage to Spain, and was in the eighth day of his illness before its nature was recognised, and removal to hospital took place. Two other cases, also in seamen, had occurred in January and February, 1903, but; with these exceptions, Glasgow was free from small-pox during the period already indicated.

In September, 1903, however, it was introduced among the model lodging-house population on the south side of the river by a worker from Talla Water-works, and for several weeks at the beginning of the outbreak no cases occurred among the general population; but, during November, and more particularly as December advanced, the general population of certain districts were invaded, and, as the outbreak extended, its direction followed the main line of preceding prevalences.

* * * * *

TABLE III.—In this table the cases and deaths in each ward are shown, together with the attack- and death-rates. The ward populations stated are the means of the annual estimates, which are based on the number of inhabited houses in 1903 and 1904. It will be remembered that in the 1900-2 outbreak, the attack-rate for the city was 2.3 per 1,000; on the present occasion it was 1.5 per 1,000. In the earlier outbreak the attack-rate in the London Road Sanitary District was 9.97; in Barrowfield, 6.46; and in Calton, 4.24 per 1,000. By the adoption of the wards as sanitary districts, those of Dalmarnock, Mile-end, and

Calton now represent generally the above-named districts, and here the attack-rate again is considerably in excess of that for the wards further distant from the hospital, or for the whole city, the figures for the wards just named being respectively 4.1, 3.7, and 1.7. Only one other ward presents an incidence corresponding to the higher rates here shown, viz, Broomielaw, where 25 cases occurred out of a population of 8,000, giving a rate of 3.04. But it should be explained that 12 of these cases were removed to hospital within one fortnight, and 10 at least were known to have been in association with a case of hæmorrhagic small-pox, which had proved fatal, and had not been recognised as small-pox until the occurrence of the disease among the patient's friends.

It must be remembered, however, that while the attack-

TABLE III.—GLASGOW: SMALL-POX, 1903 AND 1904.

Return of Cases and Deaths in each Municipal Ward.

Municipal Wards.	Mean Population.	Cases.		Deaths.	
		Number.	Rate per Million.	Number.	Rate per Million.
1. Dalmarnock, Hospital in this Ward	50,757	208	4,098	20	394
2. Calton	38,463	66	1,716	4	104
3. Mile End ...	43,174	160	3,706	11	255
4. Whitevale ...	33,432	57	1,705	5	150
5. Dennistoun ...	33,378	15	449	—	—
6. Springburn ...	41,823	37	885	3	72
7. Cowlairs ...	25,059	7	279	2	80
8. Townhead ...	39,671	33	832	5	126
9. Blackfriars ...	22,792	25	1,097	2	88
10. Exchange ...	2,235	2	895	—	—
11. Blythswood ...	3,540	3	847	—	—
12. Broomielaw ...	8,214	25	3,044	2	243
13. Anderston ...	29,297	16	546	—	—
14. Sandyford ...	26,216	31	1,182	4	153
15. Park	25,087	3	120	—	—
16. Cowcaddens ...	39,075	32	819	1	26
17. Woodside ...	45,356	26	573	2	44
18. Hutchesontown ...	41,719	73	1,750	7	168
19. Gorbals	36,550	53	1,450	4	109
20. Kingston ...	34,859	53	1,520	3	86
21. Govanhill ...	33,835	34	1,005	2	59
22. Langside ...	31,142	7	225	—	—
23. Pollokshields ...	17,358	2	115	—	—
24. Kelvinside ...	19,371	2	103	—	—
25. Maryhill ...	36,973	3	81	—	—
Institutions ...	18,509	180	9,725	14	756
Harbour	1,241	1	806	—	—
No. Residence ...	—	4	—	—	—
City	779,126	1,158	1,486	91	117

rates are here calculated over the whole population, the incidence of the disease on the section among whom the disease occurred, viz., the proportion unprotected by recent vaccination, would be considerably greater, because a very large number of those (over 400,000) who were revaccinated during 1900-2 will still form part of the present population. If, instead of calculating the attack-rate over the whole population, it had been possible to calculate it on the susceptible proportion only, it is doubtful whether there would have been any reduction in the rate at all. It is also worth remembering that the proportion revaccinated in 1901 in the districts immediately surrounding the hospital was greater than in the other districts where the disease was less prevalent; so that the greater incidence in these districts on the present occasion is all the more striking.

DR. BUCHANAN: I am under great obligation to all those who have contributed to the Discussion on my Paper, not least to those who have disputed my conclusions; and I welcome this opportunity to express my thanks.

If, with the benefit of this Discussion, I were now to re-write my Paper on the spread of small-pox by small-pox hospitals during the epidemic period, 1900-4, there would be some new matter to add, and some directions in which it would be desirable to develop its argument. I do not think, however, that I should have cause to modify what I have written. In dealing with the new matter, and with observations which have been by various contributors, I am, fortunately, able to be brief. The facts reported speak for themselves; while on the argumentative side, the contentions raised by some contributors have been in many cases replied to by others.

The introductory paragraphs of my Paper, I think, make sufficiently clear my reasons for selecting for inquiry the experience during the recent epidemic period of London, Glasgow, Liverpool and Gateshead: to which towns, with some hesitation for reasons stated, I also added Manchester. After giving the main facts as to hospital influence for each of these towns, I went on to say (p. 162):—

"Besides Manchester, there may have been other towns in the Kingdom during 1900-4 in which there has been a notable prevalence of small-pox, in which small-pox cases have been received in a hospital or hospitals so situated in regard to populous areas as to permit the matter to be tested, in which it has actually been tested by careful study, in which the facts have been exactly recorded, and

have proved negative as regards hospital influence. If such cases exist, I regret that I have not been able to find them, and consequently have not included them in my Paper."

This statement was made in all good faith, and in the hope that in the Discussion other instances, positive and negative as regard hospital influence, would be brought out, which satisfied the requirements which I had formulated, or could be utilised for scientific examination of the subject in hand. This has been the case. On the one hand, Dr. Ker has given us an interesting account of the Edinburgh small-pox hospital during 1904: negative as to hospital influence; on the other, Dr. Wellesley Harris has narrated his experience of hospital influence at Southampton; and Dr. Thresh has told us of recent distribution of small-pox in Essex in relation to the Dagenham hospital. In addition, although before the period dealt with in the Paper, we have heard from Dr. Niven the story of the relation of small-pox and the Westhulme (Oldham) Hospital in 1892-3, and the very instructive instance of hospital influence round Monsall Hospital (Manchester) in 1894.

Negative instances, in the sense of the quoted paragraph, are of the greatest value. They should be looked for, examined in detail, and considered in all their bearings along with positive instances. I need not add to the observations of Dr. McVail on this point. But I am disposed to agree with Dr. Thresh when he draws attention to the paucity of "negative instances," which can be regarded as helpful to a consideration of the "frequency" with which hospital influence has been exerted. There is no value in the negative instance if the hospital is in a desert; or if the hospital stands in the centre of a town, but has contained only an insignificant number of acute cases of small-pox at any one time; or if the facts as to house-invasion round the hospital have not been ascertained. These are extreme instances, of course, but the same considerations apply in other cases. I have been taken to task for a sentence in which I pointed out that, in several epidemics where hospital influence has been clearly manifested, the proportion of dwellings near to the hospital which have been invaded by small-pox has not been 100 per cent., but usually something much nearer 5 per cent. The reminder, however, was not wholly unnecessary. Suppose a small-pox hospital, in use during an epidemic, and containing sometimes 20 or 30 acute cases at one time, had within half a mile of it 200 dwellings and 1,000 population. I take this number so as to make the hospital

transgress the Local Government Board standards, to which so many references have been made. If during the epidemic these 200 dwellings showed a house-invasion rate of 5 per cent., or 10 houses, and this were a higher rate than elsewhere in the district concerned, there might be reason to suspect "hospital influence." The case would be worth detailed inquiry. But unless the circumstances were very exceptional, I would hesitate to conclude that hospital influence had been exerted when the figures to go upon were so small. On the other hand, if none of the 200 dwellings were invaded by small-pox during the epidemic, there would be reason to suspect a useful "negative instance." Here would be a case in which, *prima facie*, it would be reasonable to expect to have got some evidence, however slight, of hospital influence. In this case, also, the circumstances would repay full inquiry. But the hesitation in drawing definite conclusions, which would apply if the positive result had been obtained, should also apply to the negative.

I have laboured this point, because it seems to me that misuse of the term "negative instance" lies at the root of much misunderstanding on the subject: illustrated, in the Discussion, by the objection taken by Dr. Reid and Professor Kenwood to my "selection" of cases, and by Dr. Newsholme's observations (p. 186), on "high percentage of failure."

If any small-pox hospital near which there has been no small-pox is accepted as a negative instance, it is not at all surprising that Dr. Kenwood can produce one such instance to set against every case in which hospital influence has been exerted; while none will question Dr. Boobbyer's statement that the Local Government Board (who, by the way, have never attempted such an inquiry) have found no evidence of hospital influence "in the case of four-fifths of the hospitals in the country." Like Dr. McVail, I hoped that Dr. Reid (of whose evidence in the "Nottingham case"* I was aware) would give the Society some valuable data regarding his seventeen small-pox hospitals in Staffordshire. But I am still unable to put the Staffordshire experience, as narrated by him, into line with the negative instances of Manchester and Edinburgh. For an inquiry such as we have been engaged in, I do not see how to draw conclusions from aggregates of beds in hospital, from aggregates of patients in all stages of small-pox in all these hospitals together during uncertain periods, or from

* The point at issue in this case was not whether the Nottingham Hospita had or had not spread smallpox, but whether it should be closed to prevent the possibility of its doing so.

general statements that so many of the hospitals had failed to comply, as regards surrounding population, with the Local Government Board standards. I hope, however, that Dr. Reid will at a future date record in detail all the facts available with regard to some one—or still better, every one—of the hospitals in question.

Among the cases of hospital influence during 1900-4 which have been debated is that of Liverpool. It has been urged that Liverpool should be detached from the series of "positive instances" of hospital influence. The argument is that it was mere accident—no more—that there was in Liverpool an excessive prevalence of small-pox round each hospital. The fact is not disputed. But in view of the further fact that this excessive prevalence was manifested in each case in the particular period during which that hospital was receiving acute small-pox cases, of past experience of other hospitals in other epidemics, of the facts as to graduation, and of other points brought out in my Paper, I am unable to follow Dr. Hope in the argument which he presented to the Society in support of this contention. Dr. Hope's attitude in the matter is, I think, to a large extent explained by the assumption which runs through his whole argument, that Dr. Reece contended that in *every one of the houses* which were invaded by small-pox within a mile of each of the hospitals, the infection of the first case had been contracted from the hospital. I do not find in Dr. Reece's report warrant for this assumption. It has not been made, so far as I am aware, in the case of other small-pox hospitals in other epidemics, and Dr. Reece in his contribution has expressly repudiated it. But it is not surprising that Dr. Hope, by looking at the matter from this point of view, while knowing that his staff had ascertained that personal communication, independent of hospital operations, had been responsible for the infection of many persons who dwelt near the hospitals (as elsewhere in the city), came to take strong objection to Dr. Reece's conclusions.

It is unnecessary for me further to comment upon Dr. Hope's argument, or on Dr. Reece's reply: each has gone fully into the matter at issue. I would like, however, to clear up two points in my Paper regarding Liverpool. Dr. Reid drew attention to the disregard in my Paper of the opinions which had been expressed by Dr. Hope antecedent to the publication of Dr. Reece's report. But these opinions, important as we may regard them, were given in advance of the collection of facts which alone could enable us to see whether, when the Liverpool epidemic was looked

at as a whole, in relation to the operations of its several hospitals, indications of hospital influence were forthcoming. To give these facts in my Paper, I was obliged to draw solely on Dr. Reece's publication. The other point is, that by the briefness of my allusion to the figures for the Fazakerley Hospital, I have unwittingly led Dr. Hope to infer that I attach little importance to indications of hospital influence in that case. I do not think that the experience of Fazakerley, if it stood by itself, could be said to permit positive conclusions on the matter; and I note that Dr. Reece has been careful in his report to warn us of the smallness of the figures available. But the Fazakerley facts, put with the Priory Road and Park Hill facts, seem to me to be very significant.

I should add, too, that as Dr. Newsholme also expressed doubt of the reality of small-pox hospital influence in Liverpool, I have re-read his observations in the endeavour to ascertain what evidence would have satisfied him that hospital influence had been exerted there. If I interpret his meaning correctly, Dr. Newsholme would require for this purpose, that throughout the whole of this city every portion or "element" of area should have been attacked by small-pox with the same intensity as every other portion or "element;" the only exceptions being the areas round the hospitals, where the invasion rates would have evenly and progressively to increase. But this is to ask for a miracle: a method of controversy which, though not without precedent among agnostics, is hardly helpful to epidemiology. Dr. Newsholme, moreover, makes what appears to me an unreasonable demand of the spot maps given in Dr. Reece's Liverpool report. These, like any other spot maps, were published as a convenient record of facts. But they do not give all the facts which were collected and exhibited by Dr. Reece. Merely to count dots on these maps, regardless of the number of dwellings or populations dealt with, is a proceeding which may easily mislead, as Dr. Newsholme himself realises, and cannot be expected to give accurate indications, even when the results of counting are dignified by the name of "infection densities."

Notwithstanding doubts which have been expressed by some speakers as to the significance of hospital influence in particular cases, and of "negative instances" in others, it appears to be common ground that small-pox hospital influence is a definite phenomenon, causally related to the hospital, and requiring explanation. It is something apart from our experience of other infectious diseases which we ordinarily aggregate in isolation hospitals. The explana-

tion must be special to small-pox, and to cases in the acute stage of that disease. Small-pox hospital influence is not an affair which was restricted to Fulham, or to other small-pox hospitals in London, in days gone by. It has since come up again and again, in connection with many different hospitals in different places and in different epidemics. It has been manifested in various ways, but its principal characteristic has been the excessive incidence of the disease on persons dwelling in the neighbourhood of the hospital while a collection of acute cases is under treatment there, evidenced on many occasions by the existence of graduation.

Reasons are given in my Paper for maintaining that Mr. Power's theory of aerial convection not only best fits the facts brought out by recent experience, but involves assumptions which in themselves are *a priori* probable. And it will be noted that in the Discussion there has been general agreement with —at any rate, little dissent from— the proposition that the air escaping from the wards of a small-pox hospital is liable to contain an abundance of floating particulate matter; that, on occasion, and in suitable conditions of atmosphere, such floating matter may be expected to be carried for considerable distances—a mile or more—and ultimately to settle without having undergone any great degree of dispersion; and that there are reasons for believing a greater potentiality of infection to susceptible persons of particulate matter in the air of a ward containing acute small-pox cases than where the disease isolated is scarlet fever, diphtheria, or enteric fever. Dr. Bulstrode, Colonel Davies, and others have made valuable observations on several of these points. Another matter about which there appears to be little dispute is, that the theory of aerial convection affords a consistent explanation of certain ascertained characteristics of hospital influence, especially of the graduation in intensity of small-pox incidence on areas in proximity to the hospital, which has been ascertained in so many cases; and also of the manifestation of hospital influence in the case of inhabited areas which are shut off from intercourse and traffic with the hospitals by barriers of one or another kind. The main criticism which the aerial theory has received in the discussion is not that it explains too little, but that its acceptance would lead us to expect more than we know to have been the case. Dr. Newsholme, Dr. Reid, Professor Kenwood and Dr. Boobbyer, in one form or another put forward such questions as these: Why, if small-pox is conveyed aerially, do we not have more instances of it?

Every hospital in use during an epidemic of small-pox ought to show hospital influence; if aerial convection be true, a "negative instance" should never occur. If hospital influence results from particular atmospheric conditions, these, whatever they are, must, in our ever-varying climate, frequently be present in the case of any hospital receiving patients during any epidemic which lasts for many months. Then, again, what are these atmospheric conditions? Let us have a clear case. If it is wind, produce a hospital area and meteorological record, from which we can see that a S.-W. wind entails an outbreak N.-E., and so on. If it is calm, produce instances where every spell of calm has been accompanied by an outbreak round the hospital.

So far as these questions involve consideration of the frequency and significance of negative instances, I do not think I can usefully add to Dr. McVail's observations, or to what I have said above. Hospitals containing throughout an epidemic considerable numbers of acute small-pox cases, and surrounded in all quarters by inhabited dwellings, are hard to come by. Still more rare is a great concentration of acute cases, such as occurred at the M. A. B. Hospital Ships in 1901-2, and, on the aerial hypothesis, might be expected to cause spread of small-pox among persons dwelling in the vicinity even though the total population in the neighbourhood was small, and the houses limited to a small portion of the area near the hospital.

I do not see how to maintain that meteorological conditions should be the only variable quantity in these cases.* The aerial hypothesis would to me be unintelligible if it was inconsistent with the operation of numerous other factors tending, in one case to enhance, in another to diminish, or in a third to annul "hospital influence." The vaccinal condition of the population concerned is, of course, all-important. Some observations recorded in my report on the Orsett epidemic may be referred to in this connection. Dr. McVail has given considerable attention to this point, as also to the question of age, in considering the frequent escapes of inmates of institutions placed near to a small-pox hospital, of which Dr. Ker gave us a striking example in the case of Edinburgh Fever Hospital. The type of the epidemic again may be expected to have a considerable effect. Small-pox in inter-epidemic years, from some unknown circumstances affecting the life-history of the micro-organism of the disease, may be very different in

* In this matter I would invite Dr. Kenwood to apply his own contention (p. 231), "those of us.....other," to the aerial theory, instead of limiting it to the theory of undetected personal communications.

its infective ability to small-pox in epidemic times. Then, again, the physical conditions may be expected to exert a marked effect. Other things being supposed equal, we should anticipate a difference in the effects of aerial convection, between a hospital in the centre of a plain and one on the edge of a high cliff. The escape of the Hydropathic and of the Craig House Asylum during the Edinburgh epidemic may, perhaps, have been related to the fact that each of these institutions is on the opposite side of a hill to the small-pox hospital. Other conditions may easily be imagined which would modify the intensity of hospital influence, or prevent such influence from being exerted: notwithstanding the occurrence of aerial convection of small-pox matter from the hospitals.

It is easy to demand, as does Professor Kenwood, proof of exact correspondence between weather conditions and hospital influence. If such proof were forthcoming, we should be saved the necessity of debating the subject. But, as I have pointed out in my Paper, it is practically impossible to ascertain the precise conditions corresponding to a given infection which has apparently been caused by aerial convection. You cannot date back a man's infection to a particular hour; and, as a rule, the most you can get in the way of meteorological conditions is some daily record kept in the neighbourhood. All the same, certain broad conclusions of the kind which I have stated, as to conditions which appear to favour aerial convection, may be drawn from a careful study of the records available. The way to get more information on the matter would be to take a hospital suitably situated in regard to surrounding population, and then, during a small-pox epidemic, to appoint a special investigator to give his whole time to inquiring into each case of small-pox near to the hospital, as it arises—after the plan adopted by Mr. Power at Fulham—and to make simultaneously, on a pre-arranged system, a series of special meteorological observations. I wish that this could be done for as many hospitals and in as many epidemics as possible. Dr. Hope—if Park Hill Hospital is again opened for small-pox in the middle of a small-pox epidemic—or Dr. Newsholme, should the Town Council of Brighton elect to isolate small-pox in the centre of that town, might be able to give valuable assistance by arranging for some such inquiries.

Like other scientific theories which we accept and make use of, that of aerial convection has its uncertainties and ambiguities. We should like to test it further, by differential methods, in a variety of conditions, which are easy

to formulate, but seldom possible to obtain in practice. For this reason I have avoided the term "proof," as applied to aerial convention, preferring the position stated in the last paragraph of my Paper. To be adopted as a working hypothesis, aerial convection must, of course, successfully compete with other current explanations of hospital influence. Our Discussion seems to me to show that it does so. I may dismiss at once Dr. Kenwood's hypothesis that people living near a small-pox hospital are, owing to poverty and bad vaccinal conditions, especially susceptible to small-pox infection. The facts are dead against him. One of the elementary facts of "hospital influence" is the frequent persistence and repeated occurrence of excessive rates of invasion in areas near to the hospital; notwithstanding that, owing to the special local prevalence of the disease, the majority of the population of these areas has become protected from attack to a far greater degree than people living outside those areas. Dr. Chalmers emphasises this point for the neighbourhood of Belvedere Hospital. I have recorded similar occurrences at Purfleet, and in Gateshead and Felling; and there are many other instances, notably Fulham. As regards the social condition of the people living near the hospital, I may recall the inquiry which I made at Gatehead and Felling: where I got out the facts separately for each of twelve different wards of Felling and Gateshead, for comparison with the hospital area, with the results mentioned in my Paper.

It is natural to consider, as does Dr. Nash, whether flies are concerned with hospital influence. There is no doubt about the attraction which small-pox patients seem to have for house-flies. Any addition to knowledge concerning the ordinary range of flight from a dwelling of the individual house-fly, and similar matters, deserves to be considered carefully from this point of view. It must be remembered, however, that hospital influence has frequently been well marked in mid-winter, and in times of frost, and in connection with well-ordered and cleanly hospitals; in other words, at times when flies are either very scarce, or absent altogether.

There remains the explanation of small-pox hospital influence by the theory of undetected communications, which has been strongly advocated by Dr. Newsholme and Professor Kenwood. To what I have said on this subject n my Paper, I would add now a few notes.

Dr. Hamer's observation regarding small-pox ambulance traffic in London is important as a complement to instances such as the Orsett and Felling outbreaks, where ambulance

traffic as a factor in hospital influence could be altogether put out of question.

The undetected communications which Dr. Newsholme and Dr. Kenwood have in mind appear to be in large measure those between members of the hospital staff and people living in the neighbourhood of the hospital, though other communications are also assumed. Now, anyone coming with a fresh mind to this hypothesis, as an explanation of the recent cases of hospital influence referred to in my Paper, or of such cases as those reported by Dr. Wellesley Harris and Dr. Niven, would at once ask for the facts which form the basis of the assumption. We are all on common ground in knowing that person-to-person infection (in which the infectious matter has only a short journey through the air) is the common method by which small-pox is spread. We all agree with Dr. Boobbyer that the source of infection of a series of connected cases of small-pox is not infrequently missed; and that in individual small-pox cases, where there is no question of hospital influence, it is sometimes impossible to trace the origin of infection. Still, we know the other side, which is, that under modern conditions, during an epidemic of small-pox, it is usual, where there is no question of hospital influence, to be able to refer any given case back to an antecedent case. The epidemics now in question have been the subject of searching inquiries by many observers: County and Borough Medical Officers of Health and their staffs, Medical Inspectors of the Local Government Board, and others. What instances have there been in these cases where people living in the neighbourhood of the hospital have been ascertained to have, in all probability, come by their infection as a result of personal relations with hospital patients or hospital staff? Practically speaking, the answer is—None.* Dr. Newsholme's explanation of hospital influence by such communications is thus hypothesis, every bit as much as the aerial theory is hypothesis. And when Dr. Newsholme's hypothesis comes to be applied to any one of these more recent cases of hospital influence—still more to the whole evidence on the subject—the numerous and varied assumptions, which are required to fit it in with the facts known, seem to me far more improbable than any involved in the theory of aerial convection.

The nature of the difficulties to which I refer (in parti-

* I except the workmen at the temporary small-pox hospital on the Kent shore of Long Reach, 1901-1902, to whom I have referred in my Paper. Here the occurrence of personal communication with members of the hospital staff has to be considered as well as the operation of aerial convection.

cular to the assumptions needed before "graduation" can be explained) will be gathered from the observations of Dr. McVail, Dr. Niven, Dr. Bulstrode, and others, as also from my Paper. Nothing would be gained by their recapitulation, though I may perhaps draw special attention to the point raised by Dr. Niven, that these explanations involve an assumption of infectivity of "contacts," which is contrary to usual experience.

It is desirable, however, that I should comment on Dr. Newsholme's contention in regard to the Orsett outbreak and the M. A. B. Hospital Ships, and incidentally this will illustrate some of the difficulties which arise in adopting his alternative theory. First, on certain questions of fact. My inquiries in Purfleet and the Orsett Union began at a time when no more than nine cases of small-pox had occurred in Purfleet. They were continued at frequent intervals throughout the epidemic. During the whole period, I was in touch with the medical practitioner who attended practically all sick persons in the village, with the Medical Officer of Health, Inspector of Nuisances, and others, who made frequent visits to the place, and I made numerous house-to-house inquiries. As a fact, I did, with the assistance of Dr. Corbett and others, make very careful inquiry as to importation of infection from other parts of Essex or from London—and this not only in regard of the first cases in Purfleet, but also as to other cases occurring later on during the epidemic. Then, as to the alleged surreptitious visits to Purfleet of members of the staff of the Hospital Ships. Lest Dr. Newsholme's remark that "a considerable body of independent evidence exists" that such communications took place should be applied to the epidemic of 1901-2, it is well that I should be definite. From first to last, frem the beginning to the end of my local inquiries, after the publication of my Report, after subsequent correspondence, after reading the evidence given by Dr. Thresh, Dr. Newsholme, Dr. Collingridge, and others in the "Nottingham case," where this outbreak was made the subject of prolonged examination and cross-examination, and at the present day, the only suggestion of such visit known to me is that referred to by Dr. Thresh (*Epidem. Soc. Trans.*, vol. xxi., p. 103): "I am convinced that the Asylums Board have fulfilled their promise, and that there has been no personal communication between the ships and the County of Essex—save, probably, the surreptitious visit of a man to his sweetheart. The only case of this kind I can hear of has been denied by the parties concerned." There was no allegation here that small-pox in Purfleet resulted from this visit.

Leaving these matters, we may consider Dr. Newsholme's hypothesis in relation to the epidemic at Purfleet. The points to be explained there have been dealt with in my Paper. It is not so much the magnitude of the epidemic in proportion to the population—though this is important—but the continued invasion of fresh dwellings by small-pox, week after week and month after month, notwithstanding all the administrative action taken, the prompt recognition and isolation of cases, and the increasing immunity which the population of the village obtained in consequence of recent vaccination or recent small-pox. Now, suppose a series of visits to Purfleet by subordinate male members of the hospital staff, managing to get a boat and rowing nearly half a mile across the water. I imagine that even Dr. Newsholme would exclude patients, medical officers, and nurses. The first assumption necessary is that no knowledge of these visits should have been obtained by various inquirers who, unlike Dr. Newsholme, were on the spot. The next assumption relates to the way in which these visits must have been contrived. Each visitor must have selected from among the houses in Purfleet one which had hitherto escaped small-pox or general vaccination and re-vaccination. Visits of this kind must have been spread over some ten months. Towards the end of that period, when not more than forty people were left in Purfleet who could be regarded as susceptible to small-pox, the unknown visitor must have selected the dwellings of the susceptible people. The next assumption is that these visitors (who were not themselves suffering from small-pox) possessed on each occasion sufficient infective ability to occasion samll-pox among those whom they visited. I note, in passing, that any ship's porter paying a visit to Purfleet in 1901 would have been wise, in consideration of his personal safety, to exchange his uniform for his outdoor clothing.

Dr. Newsholme, however, has a second string to his bow regarding Purfleet, in the suggestion that, after all, the hypothetical visitors bringing small-pox to Purfleet did not come from the Hospital Ships, but from other places in which small-pox was prevalent. But this is to demand more remarkable coincidences than ever. These hypothetical visitors must have had some reason for thus bringing coals to Newcastle. What was it? Why should they have come to the Orsett Union, to West Thurrock, and so particularly to Purfleet? Has there been another village near London, or in the whole country, as to which there has been no question of hospital influence, where small-pox

has been dealt with after the manner practised in Purfleet, where yet small-pox continued month after month to behave in this way? Moreover, this suggestion requires either the dissociation of the Purfleet facts from the circumstances of other areas on both sides of the river, and from the occurrence of small-pox on vessels moored in Long Reach, or else the assumption of a still greater series of coincidences. The demand on our imagination is not lessened if Dr. Newsholme pulls both strings at once, and tells us that part of the Purfleet outbreak was due to unknown visitors from the ships and part to unknown visitors from elsewhere.

In the other case which is intimately known to me, that of Gateshead and Felling, the assumptions necessary for explanation of the facts by Dr. Newsholme's theory are similar as regards their magnitude and diversity, though they differ in character. So would it be with the Monsall outbreak in 1893 to which Dr. Niven referred, and other cases.

Apart from the consideration that its assumptions are much less improbable, the theory of aerial convection has the great advantage of supplying a common and consistent factor. In any case, I think the debate has sufficiently disposed of the contention that aerial convection cannot be considered as a cause of hospital influence so long as there is also a possibility that the disease may have been spread by hospital communications. I do not for a moment question that such communications may on occasion produce small-pox among persons dwelling near the hospital. But that such communications, if they occur, should be given as a reason for refusing to consider aerial convection as a simultaneous—and possibly a major—cause of the local spread of infection, appears to me to be an untenable position in the light of our present knowledge.

In conclusion, I may, perhaps, express the hope that the papers and discussion will indirectly be fruitful by encouraging investigation of further problems of aerial infection, which are now becoming of increased importance. The application of the lessons learnt from small-pox hospitals to the spread of infection caused by individual cases of the disease, to which the President and others have directed our attention, is of obvious importance, in connection with inquiries as to methods by which measles, phthisis, and infectious catarrhal conditions are spread.

N.B.—The four diagrams relating to Liverpool and Gateshead which illustrate Dr. Buchanan's Paper are reproduced from the Official Reports by consent of the Stationery Office.

PHTHISIS RATES: THEIR SIGNIFICANCE AND THEIR TEACHING.

By ARTHUR RANSOME, M.D., F.R.S.

(*Read: Friday, May 19th*, 1905.)

STATISTICS of mortality from pulmonary tuberculosis throughout long series of years are of great importance from both their etiological and their economic bearing. Their variations in successive years throw light upon possible causes of aggravation or decline, and point the way towards the measures that should be taken for the abolition of the disease.

In the present Paper the following sources of information have been used:—

Through the courtesy of Dr. Tatham, of Somerset House, I have been furnished with many statistics, derived from the Returns of the Registrar-General for England and Wales, since the year 1838; the four years, 1843 to 1846 inclusive, being omitted, as the causes of deaths were not given during those years.

Sir Stair Agnew, the Registrar-General for Scotland, has, through Mr. Hamilton, kindly provided me with the male and female phthisis-rates since the year 1871; and Dr. Matheson, Registrar-General for Ireland, has afforded me the opportunity of obtaining similar statistics for Ireland since the year 1861.

Dr. Hope, of Liverpool, Sir Shirley F. Murphy, of London, and Dr. Niven, of Manchester, have given me the figures relating to these cities.

I desire to express my cordial thanks to these gentlemen for the valuable assistance which they have given to me.*

I have projected the several sets of figures thus obtained upon the accompanying Charts, so as to show graphically their variations. The method allows us to note their rise and fall in different years, to compare the shape of their curves, their differences and their coincidences, which are

* The Editor of the *Transactions* is also indebted to Dr. Hope for much assistance in securing the reproduction of all these Charts.

thus displayed much more readily than is possible with mere lists of figures.

Chart I represents the phthisis death-rates of persons, per million of population, in England and Wales, for each year since 1838, with the exception already noted, from 1843 to 1846 inclusive.

Charts II, III, and IV give the male and female rates separately: Chart II for England and Wales, Chart III for Scotland, and Chart IV for Ireland.

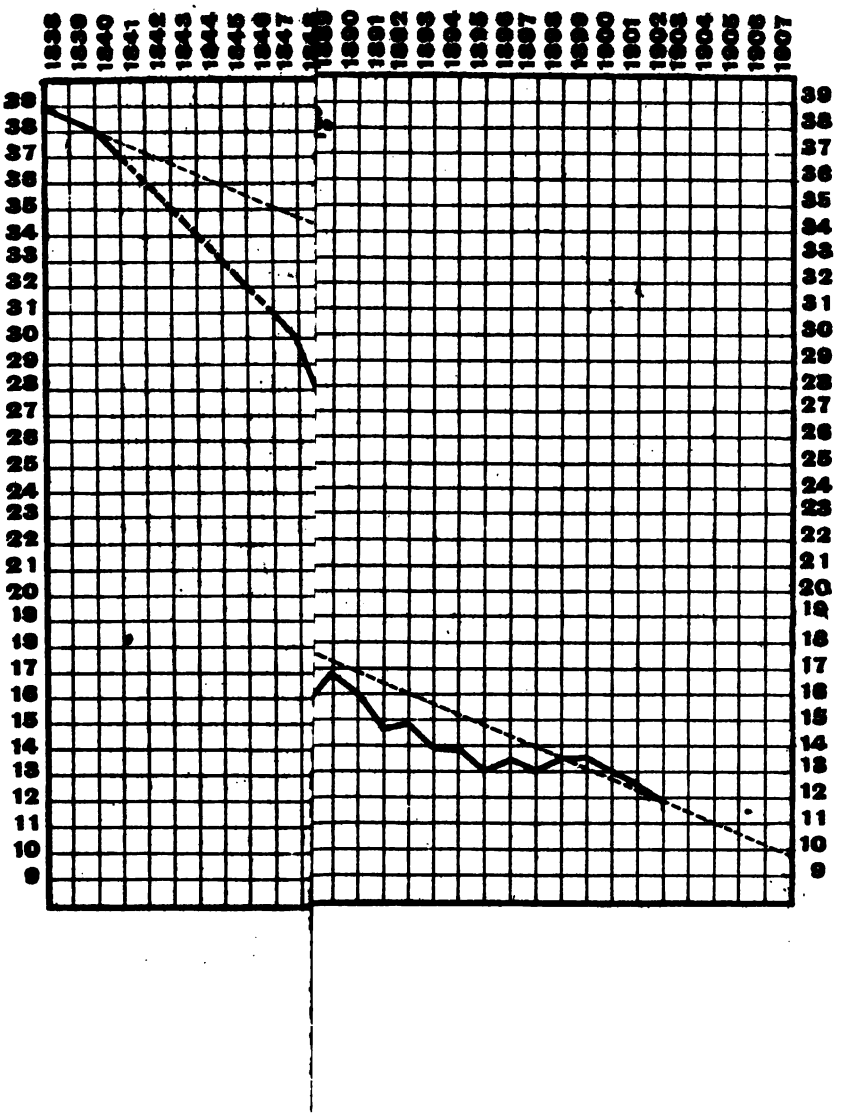
Charts V and VI supply similar rates for Liverpool and Manchester.

It must, at the outset, be pointed out that purists in the matter of statistics do not regard the figures given in the earlier years of the period thus covered as trustworthy. They prefer to limit their comparisons of the death-rates from divers diseases to the years subsequent to 1867. There were undoubtedly many imperfections in the diagnosis and in the collection of these several data in the earlier years of the Registrar-General's Returns. Thus, in his Twenty-seventh Annual Report, Dr. Farr mentions serious cases of fraud on the part of Registrars:—"In the course of twenty-nine years, out of a body of 2,200 officers, four—for the sake of a shilling an entry—inserted long series of fictitious entries of deaths which never occurred. They invented the particulars of hundreds of deaths."

Until the year 1874, the certificate of the cause of death was not compulsory. A proportion of the deaths were not certified by legally qualified medical practitioners. Dr. Farr calculated that, at one time. "in 17 per cent. of the total number of deaths, registered in England and Wales, no clue is given as to the cause of death. At one time, in South Wales, the registration books were in a state of hopeless confusion. At Llanbedr, out of 500 notices of death, 101 were certified, 399 were not; and, at St. David's, out of the same number, only fifteen were certified."

At the present time, thanks to the efforts of Dr. Farr and his successors, great improvements have taken place; but, unfortunately, even now it cannot be said that these statistics are by any means perfect; and, in the case of many diseases, they do not permit of any sound conclusion being founded upon them as to the true causes of death.

Some use may, however, be made of very imperfect statistical material: witness the remarkable researches of Dr. William Farr himself, and of Sir John Simon.


1838
1839
1840
1841
1842
1843
1844
1845
1846
1847
1848
1849
1850
1851
1852
1853
1854
1855
1856
1857
1858
1859
1860
1861
1862
1863
1864
1865
1866
1867
39
38
37
36
35
34
33
32
31
30
29
28
27
26
25
24
23
22
21
20
19
18
17
16
15
14
13
12
11
10
9

In the case of phthisis, I am inclined to think, for the following reasons, that it is not necessary entirely to reject the mortality figures during the thirty years preceding 1868.

1. Although the early diagnosis of the disease is often difficult, it must be admitted that death from pulmonary phthisis is usually quite easy to diagnose. It will, for the most part, be correctly recorded even by unskilled persons. Most old women, in fact, could diagnose the disease in its later stages quite as well as the skilled physician.

At the outset, therefore, there is a strong probability that the registration of a death from "consumption" was genuine, even if it were only reported by relations, and not by a medical man.

2. That the earlier Returns of phthisis in the Registrar-General's Reports are probably fairly correct, is shown by their accordance with what we know of this mortality from the London Bills of Mortality. In the years 1743-53, when, as Dr. Ogle says, "they were fairly accurate transcripts from the parish registers, the proportion of deaths from consumption in the Metropolis to the total deaths was rather more than one-fifth; and, in the first Return of the Registrar-General, in 1838, in London, it was 1 to 6.8."

In other words, the rate per 1,000 deaths in the former period was about 200, and in the latter about 148. Hence, in the middle of the eighteenth century, phthisis must have been still more common than in 1838; and thus the diminution in the mortality from the disease must have been proceeding steadily, at about the same rate as that observed in the earlier years recorded in Chart I.

3. A further proof of the sufficiently accurate character of the early returns of phthisis is to be found in the fact that, if a straight line is drawn from the lower end of the curve in Chart I (from 1902) to the point upon it corresponding with the year 1866, when the Returns are supposed to be fairly accurate, and if this line be then produced, it will be found to touch the highest portion of the mortality curve. The fall in the mortality from phthisis from the first year to the last is thus almost perfectly regular.

4. The male and female rates, when separated as in Charts II, III, and IV, also show a remarkable correspondence in their outlines, and testify to the general accuracy of the registration.

5. The simultaneous rise and fall of the rates in England,

Scotland, and Ireland, presently to be noticed, point in the same direction.

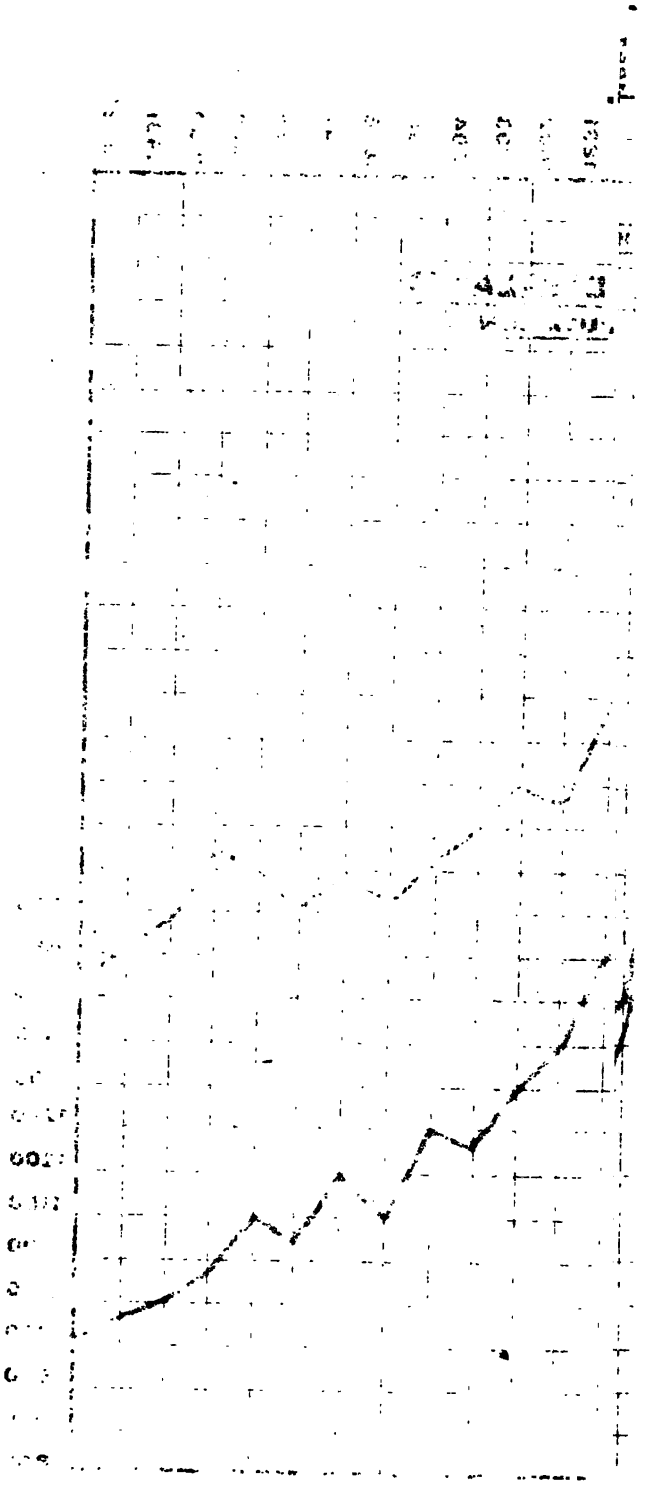
It is mainly this singular regularity in the course of the disease which makes me venture to include the early as well as the later records in my survey. There may be, and probably are, inaccuracies in the Returns for individual years; but, for the most part, these inaccuracies will be in defect, not in exaggeration of the phthisis-rate. It is more likely, for instance, that phthisis has been entered as bronchitis than that the converse has been the case. We may then, perhaps, accept the general outline of the curve as a fair representation of the truth. If this is so, and if the Charts are taken as delineating the actual course of the disease throughout the past sixty-five years, there are certain lessons which may be drawn from them.

A.—Periodic and Coincident Variations in the Phthisis-Rate.

1. The first point to be noted is a tendency to periodicity in its prevalence. Phthisis is undoubtedly a communicable disease, but it is also endemic. It clings to certain localities, and, year by year, it persistently carries off large and similar percentages from the communities in which it prevails. Unlike the acute infectious complaints, such as scarlatina, measles, etc., it shows but little variation in its exactions from year to year, and pursues a comparatively steady course. As the several Charts show, it may, on the whole, as in Ireland, rise, or it may fall, as in England and Wales and Scotland; but successive years display very little difference in the death-toll which it levies. There are, however, signs that even this chronic ailment is affected by widespread influences, such as meteorological conditions, and, possibly, that it may share with the more acute diseases the tendency to observe "periodic" times. Thus, from Chart I it appears that there were faint indications of a periodic rise in the disease in 1853, 1866, 1878, and 1890; and thus, if we begin to count from 1840, there are five periods of from twelve to thirteen years each. The indications are undoubtedly faint, but they may be sufficient to show that, as in the case of the acute exanthemata, there was, in the intervals of the periods, an accumulation of susceptible persons who had escaped the slighter infective influences, but who succumbed to the increased—almost epidemic — potency of the infection in these particular

MALE

FEMALE



PHTHISIS RATES
PER 1,000,000 OF POPU
MALE
2350
2300
2250
2200
2150
2100
2050
2000
1950
1900
1850
1800
1750
1700
1650
1891
1892
1893
1894
1895
1896
1897
1898
1899
1900
1901
1902
1903
1904
1905

years: to the καταστασις λοιμοδες as Hippocrates would have called it. This, however, is mere conjecture, and I do not lay any stress upon the occurrence.

2. It is evident, from the coincidences in the rise and fall of the disease in the three divisions of the kingdom, that there has been some widespread influence at work affecting the rate of mortality from phthisis.

(*a*) In the first place, there is a singular uniformity in the undulations of the male and female phthisis-rates in all three countries. In England (Charts I and II), they rise and fall together in 89 per cent. of the years recorded, and only 11 are opposed in the directions of their course.

In Ireland (Chart IV), 73 per cent. are in the same direction, 24 per cent. in the opposite course, and 3 per cent. are neutral. In Scotland (Chart III), 83 per cent. are concurrent, and 17 are opposed.

In by far the majority of the years, therefore, there were conditions present which simultaneously raised or lowered the mortality from phthisis in the male and female populations respectively.

(*b*) Again, on comparing the English and Scotch curves, we find that, in 95 per cent. of the years in which there was any definite rise or fall, the two curves agreed. The English and Irish curves show about 80 per cent. of agreement; the Scotch and Irish, 78 per cent. Obviously, the conditions influencing the mortality not only affected the two sexes, but they extended over the broad areas of Great Britain and Ireland.

(*c*) As might be almost assumed from the above-mentioned concurrences of the curves, the general contours of the lines are remarkably alike, especially in England and Scotland. In these countries they rise to their highest and sink to their lowest points, almost in the same years.

In Ireland the same tendency is to be observed, but owing to the general rise of the disease in that country, while it has been falling in England and Wales, it can only be noted in the lesser undulations of the curves.* We conclude, therefore, that there are widespread influences at work, extending over the whole of the British Isles,

* As I have already pointed out, these singular coincidences in the rise and fall of the disease in the three kingdoms are an additional proof of the general accuracy of the Returns.

affecting both males and females, increasing or diminishing the death-rates from pulmonary tuberculosis.

It is natural, in the first instance, to turn to meteorological conditions for enlightenment as to the causes of the coincident variations in the death-rates, but the problem to be faced is an extremely difficult one.

In any attempt to compare our figures with the several elements of meteorology, we are met, firstly, by the extreme complexity of the conditions which make up what is called the "weather" of any given year; and secondly, by the impossibility of fixing the dates of origin of the mortal disease of which the termination only is recorded in the Tables.

The origin of acute diseases may often be brought into relation with certain of the meteorological elements, owing to their prevalence on certain dates; but in chronic ailments such as phthisis, the date on which the disease was contracted is always uncertain, and the fatal issue is usually separated by many months or years from the date of origin. The period of duration of the intervening illness is also so variable in different cases, as to prevent any direct comparison between the heat or cold, the wetness or the dryness, of the year in which the complaint may have started.

It is highly probable, indeed, that certain states of the atmosphere may precipitate the fatal ending of even a chronic ailment, and that thus some slight perturbation in the course of the curve of disease might be produced which would point to its cause, if the weather returns and the death-rates were taken within sufficiently short periods of time. In this case, monthly, or even quarterly, returns would be likely to afford a basis for comparison better than annual Returns.

Through the courtesy of the Mersey Docks and Harbour Board, I have been supplied with interesting information respecting the weather in each year from 1867, and cordially thank them for the Tables now appended (Appendix I). I have, however, been quite unable to trace any definite connection between the phthisis mortality and the weather, owing probably to the reasons which I have just given; nothing in the temperature, in the wetness or dryness of the years, shows any distinct reason for the rise or fall of the disease curves. The solution of the problem must, therefore, be left to those who are better equipped for the research than I am.

B.—The Prospect of Abolishing Phthisis.

The next important lesson to be learnt from Chart I is a hopeful one. The total decline in the phthisis death-rate since the year 1838 is from over 3,800 per million to 1,100 — a fall of over 70 per cent. — more than two-thirds reduction.

In a Paper read before the Epidemiological Society in 1894, entitled "Tuberculosis and Leprosy: a Parallel and a Prophecy," I ventured to call attention to this extraordinary decline in the death-rate from phthisis, and to compare it with that from leprosy. I then pointed out that, if phthisis diminished at about the same rate during another thirty years, it would have entirely disappeared by the end of that period.

Ten years have passed since then, and, although unfortunately the rate of diminution has somewhat slackened during this time, the phthisis-rate has fallen from 1,400 to 1,100; and we need only to amend our prophecy by extending the term of existence of the disease to the year 1930.

The last dregs of the poison will perhaps be difficult to cleanse away, but, unless sanitary progress is arrested, or some adverse circumstances arise in the interval, we may surely look for the almost total abolition of the disease in England by the middle of the present century.

C.—Causes of the Decline in the English Phthisis-Rate.

When we study the course of phthisis mortality, as shown in Chart I, during the past sixty-five years, it becomes evident, from the continuous steadiness of its decline, that it has not been due to any special measures of repression, but that there must have been some influences at work acting continuously throughout the whole period. Therefore, knowing what we do of the nature of tubercular infection, we must adopt one of the following conclusions (1) that, during this period, the nation has gained increased power of resistance to infection; (2) that there has been some impediment to the cultivation or growth of the specific germ; or (3) that the microbe has been afforded fewer opportunities of attack. Probably all these several causes have been at work at the same time, and I think that we shall find that most of them may be grouped under the same head, as measures of sanitary reform.

When we look back, through the history of the last hundred years, for the first indication of a general sense of the value of sanitation, we find it most distinctly in the awakening of the nation after the terrible visitations of cholera and influenza in the years 1831, 1832 and 1833.

As Dr. Chevers says, in his treatise "On Removable and Mitigable Causes of Death," p. 40: "The cholera of 1832 certainly had the effect of awakening the medical profession and conductors of the public press to the necessity of imitating our continental neighbours in their systems of public hygienic conservancy."

Truly, as he points out, "all that was proposed and achieved in England, expressly with a view to the preservation of the public health, antecedent to the present (nineteenth) century, might be comprised within a few sentences." It is true that he was able to point to a few legislative enactments on the subject in previous years, but nearly all the great sanitary reforms have taken place since the accession to the throne of our late good Queen Victoria.

Perhaps the most important of the earlier sanitary measures after 1832 was the institution of the Registrar-General's Returns of Births, Deaths, and Marriages in 1838. The revelations made by his earliest Reports, as to the enormous waste of life then proceeding in all our large towns, brought about the Health of Towns Commission, in 1842; and, shortly afterwards, steps were taken to mitigate their unhealthiness. Several towns appointed medical officers of health, notably Liverpool in 1847, and the City of London in 1848; and other towns soon followed their example. In fact, the education of the public had begun, and a flood of literature from the pens of Dr. W. Farr, Dr. Southwood Smith, Mr. Edwin Chadwick, and others, on sanitary reform, soon took effect on the minds of the more thoughtful of the people, and the country began to attempt to cleanse its Augean stable.

In 1845, laws were passed containing regulations respecting buildings, their drainage and ventilation, and this was soon followed by others in the same direction. In 1848, the first Public Health Act was passed; also the laws against the sale of diseased meat, and other beneficent measures followed, such as the abolition of the Window Tax, in 1851.

All this activity in the cause of the public health must have had an effect, not only on the general death-rate, but also on the phthisis-rate, though no idea was entertained

at the time that this disease was at least as preventible as many others. We are now better able to understand the results of this unconscious campaign against phthisis; and it may be worth while to indicate the modes in which the several sanitary measures acted in reducing the hitherto heavy mortality from tubercular diseases.

1. *Ventilation.*—At the present day, no one can doubt the close relation that exists between "re-breathed air" and phthisis. It has been established by a thousand proofs, and need not here be elaborated. It may be explained, however, in several ways: (1) by the greater frequency in such air of the specific germ of the disease; (2) by the long life of the bacillus of tubercle in the presence of the aqueous vapour of the breath;* (3) by the co-existence of other pathogenic organisms, which prepare the way for, or hasten the action of, the specific disease; (4) by the predisposition to the disease which such air produces, either by lowering the natural resistance of the body, or by inducing diseases which open the way to the bacillus of tubercle; (5) lastly, to the fact that such air is most found where other sanitary defects, such as bad drainage and little sunlight are present; where the most delicate and otherwise diseased populations are to be found, and where intemperance and other evils are likely to assist the attack of the micro-organism.

Whatever the explanation, the Registrar-General's Reports, and the other annals of public health, teem with instances of the evil effects of respiratory impurity, especially in engendering tubercular disease, and of the immediate amelioration produced by free ventilation.

It has already been pointed out that, even anterior to the year 1832, some attempts at sanitation had been made; and among these measures must be classed the efforts, in the middle of the eighteenth century, to obtain better ventilation of ships and of public buildings.

Early in the eighteenth century, Pringle pointed out that the air of the military hospitals killed more than the sword. "Plus occidit aer quam gladius;" and he called in the assistance of skilled mechanicians to improve their ventilation. It was at his request that, in 1726, trial was made in the House of Commons of a centrifugal fan; and, a few years afterwards, we are told that as many as twenty Reports on the subject had been laid before the House. Before this, the better ventilation of the ships of the

* See p. 23, *Researches on Tuberculosis*, Weber Parkes Prize Essay, 1897, by the writer (Smith, Elder and Co).

British Navy had been carried out; and about the same time the heavy infantile death-rate at the Rotunda Hospital, Dublin, had been greatly diminished by the introduction of improved methods of ventilation. Other hospitals and public buildings soon followed these examples; and we cannot doubt that the public mind was turned in this direction, and that people were becoming alive to the danger of breathing air fouled with respiratory impurity. Probably these facts will account for at least a part of the diminished rates of mortality from consumption before the year 1836.*

2. *Land Drainage*,—Another important sanitary measure was also partially set to work in Great Britain before the great period of sanitary reform—I mean, land-drainage on a large scale. This was no doubt done with a view to the interests of agriculture; but, incidentally, it put a stop to malarial disease in the Fen country, and we may be sure that it also affected the phthisis mortality.

Extensive systems of sewerage were also established in many large towns early in the century, and sewage commissioners were appointed; and the drying of the soil which resulted, not only lowered the general death-rates of the places concerned, but, to the astonishment of many sanitarians, diminished greatly the phthisis-rates of most of the towns thus improved.

Commenting upon Dr. Buchanan's account of the results of "improved drainage and water-supply" (in 1866), Sir John Simon remarks ("Ninth Report to the Privy Council"), that "the novel and most important conclusion suggests itself, that *the drying of soil, which has in most cases accompanied the laying of main sewers in the improved towns, has led to the diminution, more or less considerable, of phthisis.*"

As we know, Dr. Buchanan continued his researches; and, in an inquiry into the distribution of phthisis on impervious and porous soils, he confirmed his observations and, without knowing it, those also of Dr. Bowditch, of Massachusetts, that phthisis is most rife on impervious, undrained soils. A few years later, the Registrar-General for Scotland also corroborated the facts as stated.

After giving a list of fifteen towns in which phthisis had

* It is interesting to observe that, about this time, Dr. Franklin, the famous American, was accustomed to take what were then called "air baths," and it was proposed to use them medicinally (*Dict. des Sciences Medicales*, Paris, 1820, Art. "Ventilation," p. 167).

thus been diminished, Sir John Simon observes: "the fact that, in some of these cases, the diminished fatality of phthisis is by far the largest amendment, if not the only one which has taken place in the local health, becomes extremely interesting and significant, when the circumstance is remembered that works of sewerage, by which the drying of soil is effected, must always of necessity precede the accomplishment of other objects (house-drainage, abolition of cesspools, and so forth) on which the cessation of various other diseases is dependent."

I do not think that this singular influence of dry subsoils in preventing phthisis has ever been fully explained; but, accepting the fact as we must, I am now inclined to trace a considerable part of the decline in the earlier portions of the phthisis-curve in England to the general and special drainage works which were actively carried out about this time.

3. *The Sanitary Improvement of Dwellings.*—The most potent of all the means which have hitherto been unconsciously employed for the suppression of phthisis is the improvement of dwellings, and especially of artizans' and labourers' dwellings.

In a paper read before the Epidemiological Society on May 11th, 1887, on "Tubercular Infected Areas," I pointed out the fact that tubercular infection clings to certain houses, and even to certain unhealthy areas; and this observation has also been made by Dr. Flick, of Philadelphia, Dr. Niven, of Manchester, and others.

Moreover, researches, pursued by myself, in conjunction with Professors Dreschfeld and Delépine, of Manchester, proved that tubercular sputum would live for months in a small, badly-drained, ill-ventilated cottage in Ancoats: whilst it was speedily deprived of all virulence in a healthy dwelling on a dry, sandy soil.

The observations of Dr. Russell, of Glasgow, and others, on the prevalence of phthisis in one-roomed tenements, as compared with the better classes of dwellings, show strongly the influence of the home upon the disease.

It would not be surprising, therefore, to find that the many Acts which have been passed since 1848 had resulted in a great diminution of the mortality from phthisis. Most large towns have also secured private Acts, by means of which "rookeries" have been abolished, unhealthy areas have been cleared, and healthy dwellings erected in their stead. Beyond all this, the Building By-laws of some

enlightened communities, such as London and Liverpool, have secured the adequate entrance of light, as well as air, into streets. Wherever all this has been done, we find a great improvement in the health of the inhabitants, and especially a smaller proportion of deaths from tubercular disease. We cannot doubt that much of the obvious decline and fall of this mortality, in the middle and end of the last century, has been due to these improvements in the sanitary conditions of the dwellings of the poor.

4. *The Amelioration of Social Conditions.*—We cannot, however, claim all this diminution of mortality as due to purely sanitary reforms. It seems certain that the resistance of the population to infection must have been increased by means of the better food, and better clothing, and more healthy environment, that became possible after the Repeal of the Corn Laws in 1847, and by the subsequent adoption of the principle of free imports into the country. We might perhaps expect, therefore, to find some evidence of this fact in a further and more rapid decline of the curves of mortality from phthisis. There is, however, nothing very decisive to note on this point. It is true that there is a somewhat rapid decline of the curve after 1847; but we must remember that food did not at once become cheaper until some little time after the cessation of the duties; and again, that most of the cases of the disease which ended fatally in 1847 must have commenced many months before that date. Moreover, there is again a rapid rise in the mortality four years later, in 1851, so that we can hardly claim the preceding descent as evidence of greater resisting power.

During the Cotton Famine, from 1863 to 1866, there is a decided rise in the curve, as if the privations of that time had been able to destroy a certain number of the more weakly individuals; but the curve again descends rapidly after 1866, thus showing that no permanent injury had been done to the workpeople.

Again, after 1870, when the prosperity of the country was said to be "advancing by leaps and bounds," there is a great drop in the mortality, but it is not possible to differentiate it from the improvement that was evidently going on owing to other sanitary reforms which have already been noticed. We must be satisfied to group all these beneficent influences together, and note their combined effect.

This is one view of the progress of social reforms which

have been favourable to the public health and hostile to the bacillus of tubercle. On the other hand, it is certain that some social conditions which are likely to add to the danger of infection by the organism have become greatly aggravated during the period in question. Thus, a much larger proportion of the population have come from country districts into towns. Most of these people, also, both males and females, have gradually drifted into the most crowded and least healthy parts of these towns; and, in place of wholesome outdoor work, they have taken to indoor occupations in, for the most part, badly-ventilated work-places, and their leisure time is usually spent in unhealthy surroundings.

Consider, for instance, the sort of life which a healthy country lad is likely to lead when he migrates into a town. He probably takes up his abode in a "common lodging-house," where his next bedfellow may be consumptive, and where the spitting habits of the whole community scatter filth and infection broadcast. He obtains work in some place where the atmosphere is polluted with similar material, and where dusts of all kinds are ready to prepare the way for the specific organism to enter his lungs and dwell there. In the evening, he perhaps goes to some dirty, badly-ventilated music-hall, or to the pit of a theatre, where again the air he breathes is contaminated with much tubercular matter; or he may go with his mates to a public-house, and sit there for hours in at least equally-unhealthy surroundings. It is, surely, not surprising that many of these young males should contract, and die of, phthisis.

The influence of the common lodging-house in spreading phthisis is very grievous. Thus Dr. Niven, in his admirable Report upon these places, presented to the Manchester Corporation in 1899, says (at p. 5): "There is one disease —viz., consumption—which does not need to be introduced, being already in possession, which plays great havoc amongst the inhabitants of common lodging-houses." From seventy-seven such houses in the Angel Meadow district, containing an average population of 1,825 persons, the death-registers furnished, in the years 1893-95, 110 deaths from phthisis, giving an annual death-rate from the disease of over 20 per thousand during that period."

"Nor is the mischief confined to the inhabitants of these houses. The consumptive labourers, hawkers, joiners, bakers, butchers, tailors, mechanics, clerks, washerwomen, will not only give the disease to their workmates who do

not live in common lodging-houses, but may in some employments convey it to those who purchase the goods they are engaged in making or vending. Moreover, they will certainly disseminate it in the district in which they live."

Take the case, again, of a female immigrant into a large town. She is pretty sure to drift into some unsanitary "slum," or into a dirty, overcrowded lodging. She takes work in a dusty, ill-ventilated factory, or she marries early and lives in a tenement-house, or in a dwelling all of whose windows are shut in order to keep out the outer air, polluted as it is with "smuts" and other impurities. Her chief amusements are gossiping in similar houses to her own, or an occasional visit to dusty, hot, and close music-halls. On the other hand, she may be devout, and may spend some time in dark and badly-ventilated chapels or churches; but in all these places she constantly runs the risk of tubercular infection. These are all reasons for an increase, instead of a diminution, in phthisis mortality; and if it were not for the enormous improvements which we have seen to have taken place of late in the sanitary condition of our large towns, the great immigration into them of young males and females must have led to this result.

As matters are now, however, the conditions of life in our country villages are often worse than those in towns, and they only escape a larger phthisis-rate owing to the smaller volume of infective material and to the sparseness of their populations.

5. *Temperance.*—The question of the relative sobriety of the populations at different epochs has from two points of view an important bearing upon our subject:—(1) Because of the predisposition to the disease produced by intemperate habits; and (2) because of the facilities for infection met with in the places frequented by drunkards.

It is somewhat difficult to estimate the exact amount of growth of temperance throughout the nation. On the one hand, from ordinary observation, it would appear that there really is less drunkeness now, in most classes of society, than there was fifty or sixty years ago. It is certainly so in the upper classes; and the movement in favour of temperance has spread widely in the Navy and Army, and, to some extent, among the working classes.

On the other hand, from Mulhall's statistics, quoted by Dr. Niven, in his paper "On the Economics of Health," it

may be shown that very little change has taken place since 1853 in the consumption of beer and spirits per head of the population; so that, if there is less drunkenness, the use of alcohol must be more widely spread among the people, and the public-house continues to be, perhaps, more than ever the "Club of the Working Man."

Of the two factors, then, one, inebriety, may have diminished; and, if so, this diminution can hardly fail to have had an influence upon the phthisis-mortality. There will now be fewer drunkards, predisposed to consumption by their habits of life; fewer drunkards' homes, in which the seeds of the disease might have been sown in the rising generation; fewer neglected children to carry on the infection.

But if, on the other hand, the public-house has not lost its hold on both the idlers and the workers of the population, then, even though there be less drunkenness, a most potent breeding-ground of the disease is left, and its influence is shown in the excess of male phthisis-mortality.

There is, however, a possible source of fallacy which should be taken into account, in our reasoning on this and other statistical points in relation to this subject.

Dr. Niven has pointed out to me the fact that, of late years, there has been a distinct alteration in the numbers of the population in the different "age-groups"; that the number of young persons at ages little affected by phthisis, *e.g.*, between 0 and 15, has diminished proportionately to the number of those at ages more prone to the disease: *i.e.*, from fifteen to forty-five. This change in the constitution of the population would naturally tend to increase the phthisis-rate per million. This simple cause will, therefore, to some extent explain the slackening of the downward tendency of the curve, but it will not cover the whole ground. If it were the sole influence at work, it would certainly affect both the male and the female rates. But, as we can see in Chart II, the female rate shows very little alteration in its rate of descent. It is the mortality among males that chiefly raises the curve of mortality of persons of both sexes in Chart I.

On the whole, I am inclined to think that, in the last thirty years, habits of temperance have not availed to stem the stream of infection flowing from the other social habits of the working classes; and that, so far, they have done but little to diminish the mortality from phthisis.

6. *Notification, Disinfection, and Isolation.*—Hitherto we have not had to record any special methods for the

repression of the disease. Phthisis was indeed recognised as to some extent a "preventable" disease, by such masters of sanitary science as Dr. W. Farr and Sir John Simon; but, beyond recommendations of better ventilation of barracks, workplaces, etc., and of the provision of lung-spaces in towns, and of better drainage of districts, no direct effort was made to prevent the communication of the disease. No such means of disinfection as was practised in Italy and the south of France, for instance, was ever used.

Consumption was indeed regarded as an infectious disease by many physicians long before the discovery of the bacillus of tubercle. It was impossible that it should have been otherwise, after the researches of Villemin, in 1865, and of other pathologists shortly afterwards. It may be safely said, however, that no practical attempts were made to prevent infection, until Koch pointed out the danger arising from tuberculous dust, and Cornet found the specific organism in the walls of rooms that had been occupied by phthisical patients.

I believe that one of the first attempts at organised voluntary notification of phthisis and disinfection of houses in England was made at my suggestion by the medical staffs of the Consumption Hospital and of the Royal Infirmary in Manchester, in the year 1892. At a meeting of these gentlemen, which was also attended by Dr. Tatham, then Medical Officer of Health for Manchester, a scheme was drawn up, and, thanks to the public spirit and energy of Dr. Tatham it was at once put into practice, and was continued for several months. It was then abandoned for a time, in order that Prof. Delépine might make experiments upon the efficiency of the methods of disinfection employed. The work in Manchester was afterwards independently and thoroughly carried out by Dr. Niven, upon a system of his own, originally formulated in Oldham, but extended and revised to meet the requirements of the Manchester City Council. About the same time (in 1893-4) an admirable system of dealing with infected persons and infected houses was carried out in New York, and various towns in England and Scotland followed these examples.

In 1895, the National Association for the Prevention of Tuberculosis was established under the Presidency of the Prince of Wales; and it has done much to call public attention to the danger of infection, and to the means of warding off the disease.

During the last few years, also, numerous sanatoria for

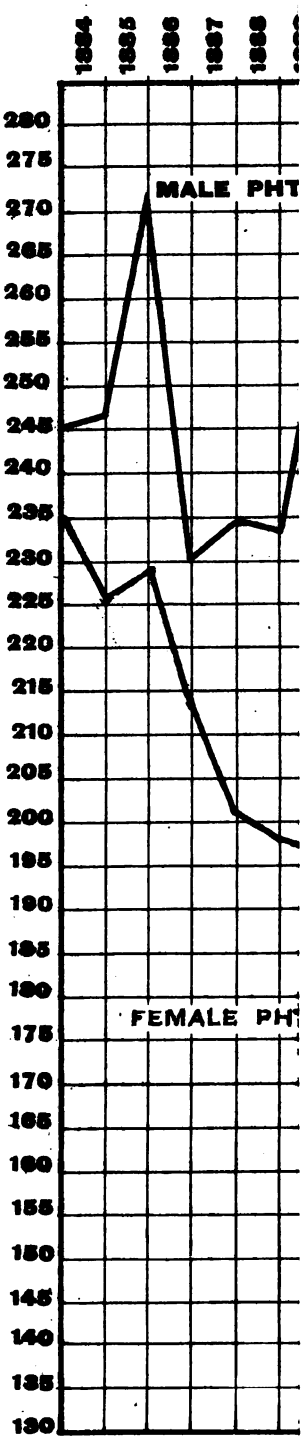
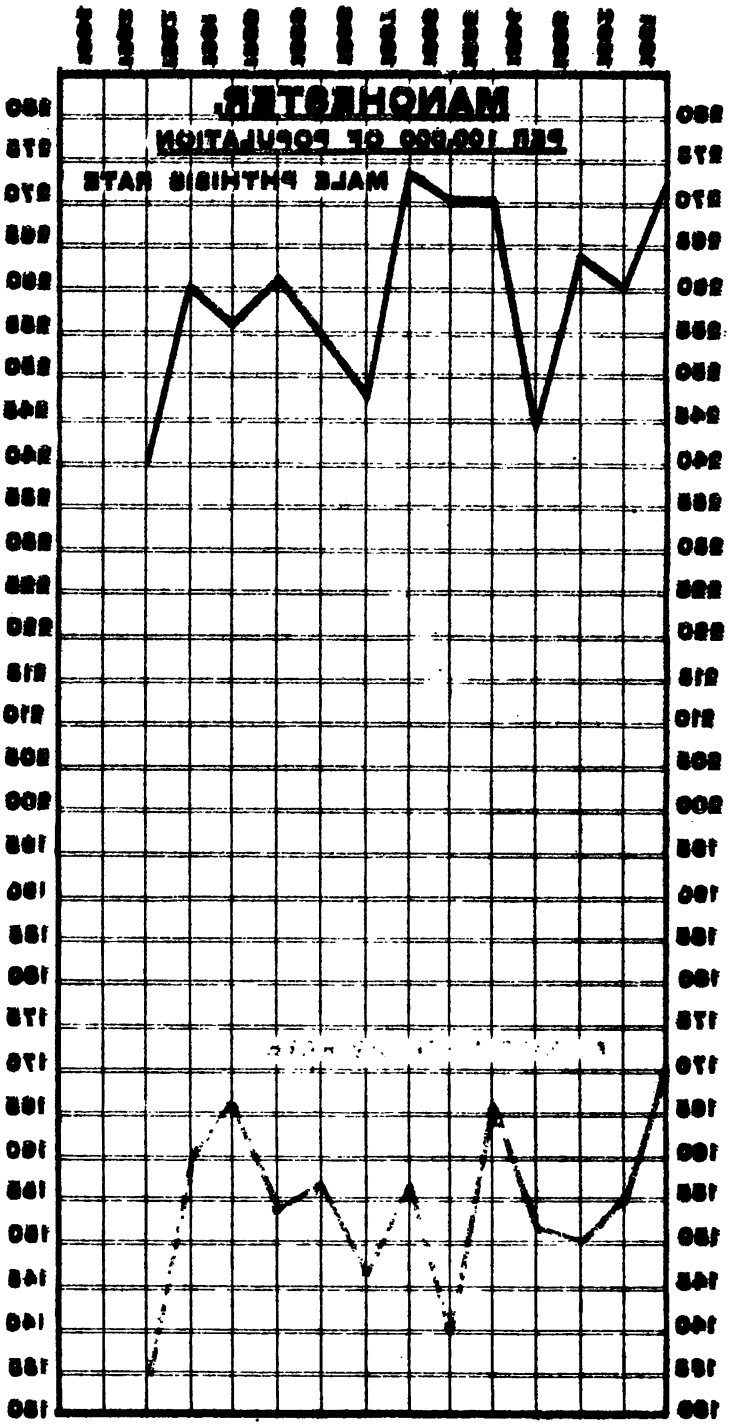

1994
1995
1996
1997
1998
280
275
270
265
260
255
250
245
240
235
230
225
220
215
210
205
200
195
190
185
180
175
170
165
160
155
150
145
140
135
130
MALE PHT
FEMALE PHT

CHART VI.



the reception of consumptive patients have been established, and their reports claim a large measure of success in combating the complaint. Altogether it may be said that, within the last decade, a fairly strong direct effort has been made to abolish consumption.

It might have been anticipated that such an active campaign against the disease would result in some noteworthy reduction in the phthisis mortality. It is disconcerting, therefore, to find that although the curve of the disease still declines on the whole, yet it falls at a diminished rate. There is even a slight rise from 1896 to 1900, and in the latter year its rate is nearly as high as it was in 1895. It falls again in the three following years, but no faster than it did from 1890 to 1895. As I have already pointed out, also, this slackening in the rate of descent cannot be due to the alteration in the grouping of the population, as it is confined to the curve of the male mortality. On the whole, I think that it must be admitted that there is something to be explained in regard to this apparent arrest in the fall of the phthisis-rate.

It cannot be supposed that the direct measures of repression which we have just passed under review have had no effect; nor can we admit that there has been any slackening in the energy with which general sanitary reforms have been carried out. I have made careful inquiries on this point in London, Liverpool, and Manchester, and, in all these places the female rates have been steadily diminishing, especially in Liverpool (Chart V), where most active work of reconstruction of unhealthy areas has been going on for many years. The male rate always lags behind that of the females, and is throughout the greater.

From returns kindly furnished to me by Professor Hope, of Liverpool, and by Dr. Niven, of Manchester, I have drawn the Charts (Nos. V and VI)—which show the variations in the male and female phthisis-mortality for a number of years. In each of these cities I am informed that from 6,000 to 7,000 unsanitary dwellings have been dealt with.* The power of sanitary reform, then, in miti-

* Since the reading of this Paper, Sir Shirley F. Murphy has kindly furnished me with the male and female phthisis-rates for London, from 1851 to 1903. These figures are uncorrected for institutions, but are sufficiently correct for our purpose, as will be seen by comparing the uncorrected with the corrected rates for the years 1895 to 1903. Time does not permit charts to be made from these figures, but they are given *in extenso* in an Appendix. They will clearly show that similar conditions to those in Liverpool and Manchester are found in London. There is the same preponderance of male over female deaths. The descent of the female rate is also more decided and more rapid than that of the males.

gating the disease among females is therein made clearly evident, but among males it is obvious that it has had less effect.

From the consideration of these facts we are almost driven to conclude that some malign influence has intervened to prevent both the sets of agencies mentioned from having their full effect upon the males of the population. In other words, that there must be some permanent sources of infection which, at the present time, are out of reach of these beneficent agencies. Now, when we look around for sources of infection mainly affecting males, there are three which stand prominently forward:

1. The imperfect ventilation of many of the work-places in which both sexes are employed, but in which the spitting habits of the males expose them to greater danger than the females.

2. The habitual use of common lodging-houses, which is more prevalent among men than women. This source of danger has already been alluded to.

3. The constant resort of the working-man to the public-house, and his long-continued sitting and inhaling of the sputum-laden air.

I have already mentioned this last source of infection as a cause of the still untouched residuum of tubercular disease. We shall find further grounds for this opinion in the next section.

D.—Causes of the Differences between the Phthisis-rates of England, Scotland, and Ireland.

Hitherto, in our contemplation of Charts II, III, and IV, we have dwelt mainly upon the singular coincidences to be found between the minor undulations of the curves. We have now to consider their equally remarkable differences and their significance.

These differences are chiefly three:—

(1) The deplorable fact that, whilst in England and Scotland the phthisis mortality has greatly diminished, in Ireland it has increased by about one-fourth, in the course of the thirty-seven years recorded by the Registrar-General.

(2) We have the strange circumstance that, whilst in Scotland and Ireland the female rate exceeds the male, in England the reverse is the case.

(3) In both England and Scotland, the female rate descends more rapidly than the male.

These facts need explanation; and in attempting to account for them, I think that we shall find some corroboration of the conclusions at which we have arrived as to the variations in the English phthisis-rate.

With regard to the increased phthisis mortality in Ireland, it is most probable that it is due mainly to the absence—in the greater portion of the country—of those beneficial sanitary reforms which we have found to be such potent weapons against tubercular disease.

Although in one or two towns a certain degree of sanitary effort has been made, it is incontestable that all over the country sanitation, such as we have been accustomed to in England and Scotland, has not been carried out. The greater part of the country is still undrained; the hovels in which the peasantry used to live and die are hovels still; cleanliness is rare, and nuisances of all kinds abound to almost as great an extent as they did formerly. In Dublin and many other towns, there are an abundance of breeding-places for the tubercle bacillus.

The Report of the Royal Commission on Towns in Ireland, in 1877, says that: "revelations of a very startling and shocking nature were brought before us, indicating an almost culpable degree of apathy on the part alike of the sanitary authorities and the inhabitants" (quoted by Sir J. W. Moore, in an Address on State Medicine in 1885).

Sir J. W. Moore also tells us that "the Sanitary Act of 1877 was so faulty and so stingy in its provisions for the payment of the Medical Officers of Health, that sanitation in Ireland has received a check, which, even if public opinion is gradually brought to bear upon the subject, twenty years will scarcely remedy" (*Dublin Journal of Medical Science*, vol. lxxix, p. 212).

We can hardly doubt that this neglect of sanitary reform has withheld from Ireland many of the beneficent measures which in England have proved so powerful in restraining tubercular disease.

We must add to these adverse influences the fact that owing to the excessive emigration from Ireland of many of the most able-bodied and healthy persons of both sexes, a large proportionate residuum of weakly and tubercularly-susceptible individuals have been left behind, to run the gauntlet of infective agencies, fostered by the neglect of sanitary measures.

Here again, however, we must point out that this influence would affect both males and females.

It would also surely have had most effect in the early years of the period, when the exodus of the population was most manifest.

It has further been suggested to me by Mr. W. J. Crossley, that some part of the increased mortality from phthisis in Ireland may be due to the fact that now buttermilk is much less used in the ordinary diet of the peasant than it was formerly.

The "Creameries" that have been established throughout the country take most of the milk, and export it in various forms, so that this source of nourishment has been greatly diminished.

On the whole, then, we must conclude that the main causes of the rise in the Irish phthisis-rates of both males and females are to be found in the want of proper ventilation of dwellings, in the lack of land drainage, in the want of healthy environment, and especially the dampness of the surroundings; in the lack of proper nourishment, in habits of intemperance, and, lastly, in the imperfect manner in which the direct means of combating the infection have been carried out. Many of the above-mentioned causes of the disease would fall most heavily upon the female population, and hence probably arises the predominance of the female over the male rates of mortality.

The adverse influences mainly affecting males in England have already been indicated, namely, the practice of spitting in ill-ventilated workplaces, common lodging-houses, and the public-house. Owing to these causes, while the female rate has been lowered more than one-half in twenty-eight years (from 2,113 to 993), the male rate has gone down less than one-third (from 2,296 to 1,427). But another point has also to be noted, namely, that throughout the whole period for which the figures are available in England, whilst the above-mentioned adverse circumstances have been able to keep the male rate above the female rate, a complete contrast to this state of affairs is to be found in Ireland and Scotland; in these countries—except at the very end of the period—the female rate is the higher of the two. It would be interesting if some explanation could be given of this difference. Probably some part of it is due to the different degrees to which the several populations are attracted to indoor employments.

Dr. Greenhow was the first to point out (in 1868) that,

in proportion as workpeople were occupied in indoor employments, so did the mortality from lung disease increase; if more males than females were so employed their rate was highest, and *vice versâ*. His observations on this point have been amply confirmed by others.

According to this rule we should expect the female rate to be usually the higher, as women more commonly work indoors, often at home, in household occupations. This is so even in England, in the agricultural counties, where the female rate of mortality from phthisis exceeds the male rate. But in this country, so great is the power of the adverse circumstances which have been mentioned, that, as we have seen, the total phthisis-rates of the males constantly exceed those of the females, and their grade of descent is less abrupt. In Ireland and Scotland, however, where the men mostly work out of doors, it is the women who suffer most from the disease. Are, then, the malign influences less potent in these countries than in England? An inquiry into the differences in the habits of the several populations might throw some light upon this question.

Looking broadly at the matter, it seems clear that there are certain differences in some of their habits. Thus, in Ireland and Scotland, although the peoples are for the most part of different religions, yet they are, especially the females, more inclined to attend divine service in church or chapel than are the bulk of English people; and these places of worship are notoriously badly ventilated. On the other hand, the English, especially the males, are more inclined to all kinds of social gatherings in equally badly-ventilated rooms. Again, although the spitting habit may be equally common among the males of all three countries, this source of infection is more likely to affect the Englishman than either the Scotchman or the Irishman. There is but little desecration of church or chapel by spitting on the floors, the women using their handkerchiefs for this purpose; but it is notorious how general is this disgusting habit in most of the places frequented by the English working-man.

In each of the three sources of infection which we have mentioned as the cause of the excessive mortality of the English workman, it is the tuberculous dust spread by this habit which is the main agent at work: in the workplace, in the common lodging-house, and in the public-house.

Although I am not well acquainted with the habits of the Scotch or Irish in this respect, yet I should suppose that it would at least be less hurtful to them than it is to the Englishman.

Owing to his prevailing occupations, the Irishman and the Scotchman would mostly spit out of doors; the Englishman indoors on the floor of any place where he might be. The drinking habits of the three nations may also tend towards this result. Although they may, on the average, take about the same amount of alcohol per person, they take it, for the most part, in different fashions. The Scotchman goes into his "dram-shop," quickly drinks down his dram, and goes out again immediately. The Irishman often does the same, though he may perhaps linger for a longer time, taking his porter or his "toddy" in sips; but we know how long an English labourer sits in the bar or tap-room, drinking successive glasses of beer, treating his friends, and discussing the affairs of his parish or of the nation. A tuberculous person spits as freely upon the floor as his healthy neighbour, and the tuberculous matter dries up amongst the sand or sawdust, gets ground into a fine powder, and when swept up flies into the air on wings of epithelium, and so is ready to lodge in any susceptible person's lungs. I believe that these differing circumstances will account for most of the differences in the phthisis-rates of the three kingdoms.

E.—The further Campaign against Phthisis.

There are several lessons to be learnt from this review of phthisis mortality, especially in regard to the future campaign against this disease.

Thus (1), although so far the direct measures of repression, such as notification and disinfection, have been overweighted by adverse circumstances, and have made no apparent impression upon the rates of mortality, yet we cannot doubt that ultimately they must have an effect, and must be energetically persevered with, especially the disinfection of houses in which phthisical patients have lived or died.

But (2) we cannot forget that, however closely the net of notification may be drawn around the known consumptives, a large number will nevertheless escape through its meshes, and will spread the specific infection broadcast.

Accordingly, it will always be necessary to use the other means of quelling the disease which have proved so efficacious in the past. We must, therefore, persevere with all the measures of sanitary reform, which act by removing the chief conditions under which alone infection becomes possible. We have seen that even when the seeds of this infection were present in large quantities, yet free ventilation, good drainage, sunshine, and abundance of good food and clothing, have reduced the phthisis mortality of males in England by nearly two-thirds, and of females by nearly three-quarters. It would, therefore, be folly to give up the weapons which have already produced so magnificent a result, and trust only to the continental methods of isolation and disinfection.

Much as has been done, much more yet remains to be done, in the way of opening up the blind streets and alleys in our large towns, the destruction of insanitary property, the building and proper management of healthy dwellings of the poor, and the preservation of as pure an atmosphere as possible in all communities of human beings. More persistent efforts will also have to be made to obtain herds of tubercle-free cattle throughout the country. When all this is accomplished, however, I fear that there will still remain the adverse influences which we have seen to weigh so heavily, especially upon the male phthisis-rates.

If we are to clear away the last dregs of the disease from the populations of these islands, it will be necessary to adopt stringent measures against the spitting habit in all public places, and also in workplaces, common lodging-houses, and public bars and taprooms. Moreover, efficient ventilation must be established by law in all these places, and also in all places of public assembly: in theatres, concert-rooms, churches and chapels; and, not only air, but adequate light must also be admitted. The education of the public, and especially of the children, in general hygiene and in the best methods of avoiding tuberculosis infection, must be more adequately carried out. When all this has been accomplished, we might then hope to see a steady and more rapid descent of the phthisis mortality curve, and might expect that, after not many decades of years, this opprobrium of our race will cease to exist.

The Appendices to this Paper will be found on pages 282 to 284, and the Discussion follows on page 285.

APPENDIX I.—MERSEY DOCKS AND HARBOUR BOARD.

LIVERPOOL OBSERVATORY, BIDSTON, BIRKENHEAD.

General Character of the Weather for the Years 1867 to 1903 inclusive.

Year.	Rain.	Cloudiness.	Temperature.	Wind.	
				Velocity.	Direction.
1867	Below average	Cloudy	Average	Below average	S.E. and W.
1868	Below average	Very bright	Above average	Above average	S.W. and N.W.
1869	Above average	Bright	Average	Average	S.W. and N.W.
1870	Much below average	Very bright	Below average	Below average	S.W. and S.E.
1871	Below average	Average	Average	Average	S.E. and S.W.
1872	Abnormal, 45.664 ins.	Cloudy	Above average	Average	S.E. and S.W.
1873	Much below average	Cloudy	Average	Above average	S. and S.W.
1874	Below average	Cloudy	Average	Above average	S.W. and N.W.
1875	Average	Cloudy	Average	Much below average	S.E. and S.W.
1876	Slightly above average	Bright	Average	Above average	S.E. and S.W.
1877	Much above average	Very cloudy	Average	Average	S.E. and S.W.
1878	Little below average	Cloudy	Average	Below average	S. and N.W.
1879	Average	Very cloudy	Much below average	Above average	S.E. and S.W.
1880	Above average	Cloudy	Average	Much below average	S.E. and S.W.
1881	Above average	Cloudy	Below average	Above average	S.E. and S.W.
1882	Above average	Fairly bright	Average	Above average	S.E. and S.W.
1883	Below average	Cloudy	Below average	Average	S.W. and N.W.
1884	Below average	Very bright	Above average	Much below average	S.E. and S.W.
1885	Average	Bright	Below average	Slightly be ow average	S.E. and S.W.
1886	Above average	Fairly bright	Below average	Much below average	S.E. and S.W.
1887	Much below average	Very bright	Below average	Slightly below average	S.W. and N.W.
1888	Much below average	Very cloudy	Below average	Above average	S.E. and W.
1889	Little below average	Very cloudy	Below average	Average	S.E. and W.
1890	Little below average	Very cloudy	Below average	Above average	S.E. and S.W.
1891	Above average	Very cloudy	Below average	Average	E. and S.W.
1892	Above average	Very cloudy	Below average	Above average	S.E. and W.
1893	Above average	Very bright	Above average	Above average	S.E. and W.
1894	Little below average	Fairly bright	Above average	Average	S. and W.
1895	Little below average	Fairly bright	Below average	Above average	S.E. and W.
1896	Little below average	Cloudy	Slightly above average	Average	S.E. and W.
1897	Average	Cloudy	Average	Above average	S.E. and N.W.
1898	Below average	Cloudy	Above average	Average	S.S.E. and S.W.
1899	Below average	Fairly bright	Slightly above average	Below average	S.E. and W.
1900	Above average	Very cloudy	Slightly above average	Above average	S.E. and S.W.
1901	Above average	Fairly bright	Average	Average	S.E. and W.
1902	Below average	Cloudy	Below average	Above average	E. and N.W.
1903	Above average	Cloudy	Average	Above average	S.E. and N.W.

APPENDIX I.—*Continued.*

Statement of Mean Maximum and Mean Minimum Air Temperatures taken at 9 A.M. *daily from* 1867 *to* 1903, *inclusive.*

Air Temperature.			Air Temperature.		
Year.	Mean Maximum.	Mean Minimum.	Year.	Mean Maximum.	Mean Minimum.
	Deg.	Deg.		Deg.	Deg.
1867	54.7	43.2	1886	53.1	43.3
1868	57.4	44.9	1887	53.4	42.6
1869	55.5	44.1	1888	52.8	42.7
1870	54.9	43.1	1889	50.4	43.8
1871	54.9	43.2	1890	53.9	43.7
1872	56.0	44.4	1891	53.6	42.9
1873	54.0	43.5	1892	52.6	42.4
1874	54.9	44.0	1893	56.7	45.0
1875	54.2	44.3	1894	55.1	44.4
1876	54.5	43.9	1895	53.4	42.6
1877	54.2	44.2	1896	54.6	44.1
1878	54.4	44.6	1897	54.6	44.1
1879	50.9	41.7	1898	55.5	45.1
1880	54.2	44.1	1899	55.2	44.2
1881	52.8	42.5	1900	54.3	44.4
1882	54.4	44.4	1901	54.3	43.7
1883	53.9	44.0	1902	53.1	43.3
1884	56.4	44.9	1903	53.9	44.1
1885	52.8	42.9			

APPENDIX II.—ADMINISTRATIVE COUNTY OF LONDON.

(SIR SHIRLEY F. MURPHY.)

PHTHISIS.—*Death-Rates per* 1,000,000 *living*, 1851 *to* 1903.

Year.	Males.	Females.	Year.	Males.	Females.
1851	3.459	2.538	1880	2.705	1.839
1852	3.330	2.566	1881	2.687	1.846
1853	3.462	2.603	1882	2.714	1.801
1854	3.337	2.510	1883	2.749	1.857
1855	3.410	2.648	1884	2.701	1.798
1856	3.152	2.519	1885	2.536	1.712
1857	3.229	2.416	1886	2.571	1.663
1858	3.162	2.420	1887	2.363	1.541
1859	3.329	2.436	1888	2.275	1.438
1860	3.224	2.417	1889	2.371	1.436
1861	3.224	2.359	1890	2.622	1.591
1862	3.177	2.380	1891	2.480	1.512
1863	3.110	2.376	1892	2.320	1.415
1864	3.474	2.482	1893	2.340	1.439
1865	3.419	2.476	1894	2.179	1.283
1866	3.705	2.589	1895	2.219	1.374
1867	3.446	2.407	1896	2.159	1.296
1868	3.411	2.386	1897	2.242	1.281
1869	3.284	2.331	1898	2.274	1.275
1870	3.349	2.275	1899	2.378	1.370
1871	3.116	2.256	1900	2.270	1.224
1872	3.118	2.121	1901	2.145	1.186
1873	3.042	2.163	1902	2.089	1.107
1874	3.000	2.034	1903	2.011	1.098
1875	3.152	2.174			
1876	3.112	2.139			
1877	2.944	2.041			
1878	3.054	2.156			
1879	3.001	1.995			

PHTHISIS : *Death-Rates per* 1,000 *living.*

Year.	Death-rate* not fully corrected for Institutions.	Death-rate† fully corrected for Institutions.
1895	1.83	1.77
1896	1.73	1.68
1897	1.78	1.71
1898	1.79	1.72
1899	1.90	1.82
1900	1.79	1.73
1901	1.71	1.66
1902	1.64	1.60
1903	1.60	1.55

* Including deaths of Londoners in the Metropolitan workhouses, hospitals, and lunatic asylums outside the County of London, but excluding deaths of non-Londoners in the Willesden Workhouse, the London Fever Hospital, the Metropolitan Asylums Board's Hospitals, and the Middlesex County Lunatic Asylum, within the County of London.

† These death-rates are fully corrected for institutions, *i.e.*, by the exclusion of deaths of persons not belonging to but occurring in institutions situated within London, and by the inclusion of deaths of persons belonging to London, but occurring in London institutions situate outside the Administrative County.

Discussion on Dr. Ransome's Paper.

The President: Dr. Ransome has added one more to the many valuable contributions he has made to the *Transactions* of this Society, and one more claim upon our thanks as members of it. I will now ask Sir Shirley Murphy to open the discussion.

Sir Shirley Murphy: I wish to express my very warm appreciation of the value of this Paper. It is a most excellent *resumé* of the subject, dealing as it does with the causes of the remarkable diminution of phthisis which we have all been watching during recent years. I confess that, whilst the general fall in the rate of mortality has interested me very much, it is really of less interest than the difference between the male and female mortality-rates. The fact that the women are deriving benefits in greater degree than men certainly does want explanation, and I can imagine various hypotheses that might tend to explain it, one of which I will offer to Dr. Ransome. In recent years, as Dr. Ransome has pointed out, the country has gone through a period of improved prosperity, and the people are better housed and better fed at the present day than they were fifty years ago. Now, when a family is poor, who is it that suffers most? It is invariably the woman who goes without; often, indeed, she starves herself. She feeds her husband, she feeds her children, but she herself will go without. But with improved prosperity she comes in for her share; and I think in all probability the people who have derived the most benefit from the improved conditions of recent years have been the women; that is to say, they have had less necessity to starve themselves. The man, who was the better off years ago, has continued to be still better fed, but the change which has come about has made very much less difference in the case of the men than in the case of the women. That is a hypothesis which may be suggested: what it is worth I do not pretend to say, but it might explain some of the differences between the male and female curves.

What are the actual circumstances that are leading to the improvement in both sexes? I know that if the sanitary areas of London are arranged, first, in order according to the amount of overcrowding, *i.e.*, the proportion of their population overcrowded; and, second, according to their rates of mortality from phthisis, you will find much paralellism between the two. That demonstrates

pretty clearly that the phthisis death-rate of a district or a town stands in close relation to the proportion of its population which is overcrowded.

Of course, one knows that overcrowding is, after all, only an expression for poverty. People who are pretty well off not only live in houses where they have more accommodation, but they are able to buy more food, and in greater variety. So that we cannot say it is the want of house accommodation, or the want of food alone, since these things are only a general indication of the social condition of the population. The social improvement which has taken place probably acts in two ways: it enables people to get more space, and it enables them also to get better food, which no doubt makes them more resistant to invasion.

Dr. Ransome has been obliged to ignore in his Paper one subject which may be related to the amount of prevalence of phthisis. I mean the occurrence of tuberculous disease in the lower animals, especially in the bovine animal. Whatever part tuberculous milk plays as an original factor in the production of phthisis, it would appear that prevalence of phthisis is more largely governed by other conditions, because the very poor, who suffer most from phthisis, are the people who get the least milk. The most important conditions may be those which tend to make the human subject resistant to the disease. If that is so, it must be largely in the direction of the improvement of social conditions that we shall have to look for the further reduction of phthisis.

Dr. Ransome has mentioned also the effect of alcohol in making people susceptible to phthisis. Apart from the deleterious action of alcohol on the tissues, it may be that alcohol replaces a wholesome food supply, and that it is in this way that the mischief is done. One knows that in many families a very large proportion of the weekly wage goes in alcoholic drink, and I can quite believe that, even with an improved state of prosperity, when wages are higher, such a large proportion of those wages goes in alcohol that the better food that the money might buy is not consumed.

Dr. Ransome has drawn attention to the fact that the decline is not as rapid as it was. I think this is also explicable on the hypothesis that better feeding makes people resistant to phthisis. I mean, that as the people have been provided with a better food supply, they have acquired all the resistance from that change that they can get; and

perhaps, more food and better food still would not have such effect as in the past in making them more resistant to the disease. I should think, in all probability, the rate of decline will get less and less; and I do not myself anticipate that in thirty years this country will be free from tuberculosis. However, some of us will not live to see that.

DR. NEWSHOLME: I should like to add my tribute of thanks to Dr. Ransome for the admirable *resumé* of our knowledge as to phthisis which he has given us; and for the indications which he has given us also for future work along lines offering good prospects of still further reduction of phthisis mortality.

Possibly Dr. Ransome has made a little too much of the slackening of the reduction in the rate of mortality from phthisis; or rather, he has expressed too strongly his sense of disappointment that the direct measures which have been taken against phthisis have not been followed by more definite results than have followed them. As a matter of fact, it is only fair to say in deprecation of that view, and in defence of those more active direct measures of notification, disinfection, and sanatorium treatment, that they have not been adopted to any considerable extent; indeed, only to a ridiculously small extent in proportion to the total amount of phthisis in the country. I think that is a point which it is necessary to emphasise. With the exception of Manchester, Sheffield, and Brighton, and possibly one or two other places, little or nothing has been done in regard to the notification of phthisis and the subsequent measures which follow upon such notification. I speak with a sense of responsibility in making that statement; for although it would be very easy to compile a list of forty or fifty districts, including metropolitan boroughs, which have voluntary notification of phthisis, in the vast majority of them (with the exceptions I have made) it is to all intents and purposes either a ridiculously small affair or a dead letter. In my own town, I am glad to say, the notification of phthisis has been a great success. I am somewhat proud of the fact that, during the last twelve months, we had a larger number of voluntary notifications than Sheffield had during the first year of its compulsory notification of phthisis. Doubtless, in Sheffield the number will increase as the compulsory powers become better known. No doubt that is partly due to the fact that when the cases have been notified we have, in

Brighton, been able to do something for them: we have been able to take them into a Sanatorium, and give them treatment. A considerable impetus has thus been given to notification.

It is certain that notification has not had more than a very scanty scope so far; and therefore one cannot wonder, even though it was accompanied by the utmost measures that could follow upon it, that the total effect on the phthisis of the country has been small.

I was greatly interested in what Dr. Ransome said about the relative trustworthiness of mortality figures for phthisis in the early years of civil registration. I think that point is open to doubt; but still I am very glad to hear his authoritative statement upon it. It certainly must appear to everyone extremely remarkable that during the early years of registration—from 1838 onwards—there was a much more rapid decline than in the later years during which that sanitary reform, to which Dr. Ransome ascribes the reduction, was very much more active.

I was also greatly interested in what was said about the periodicity which is shown in some of the English curves. The last peak (that for 1890) coincides with the first great year of the recent epidemic of influenza in this country. You will remember—indeed it is a notorious fact—that influenza hurries people suffering from phthisis into their graves much more rapidly than they would otherwise go; and I think that is the probable explanation of that particular peak. I am not able to give an explanation of the other periodical peaks; but it would be interesting to compare the periods of other epidemics with the years of increased rate of phthisis.

A point has been made by some of the fact that, although phthisis is an infectious disease, it does not show epidemic peaks as other infectious diseases do. I think it is unreasonable to expect that it would do that, because, in the case of phthisis, we have to deal with a chronic infectious —not an acute infectious—disease. And that leads me to an interesting point as to what is the average duration of cases of phthisis. It is usually stated as three years, but my own experience is that it is considerably longer than that. In my own town I have computed the amount of sanatorium accommodation required on a basis of four years' average duration of the disease; but, personally, I think even that is too short.

With regard to the slackening of the rate of decline of phthisis, I think Dr. Seaton would tell us that we must

compare that with typhoid fever, in which there has been a similar slackening in the rate of decline of mortality: the major causes of the disease having disappeared, and the minor causes, which produced scattered and sporadic cases, still remaining. We are much more impotent to deal with the causes of these minor outbreaks of enteric fever than with the causes of the major outbreaks. I think there is an analogy between that and the facts relating to phthisis. A good many minor factors in the spread of phthisis remain, and for that reason it can scarcely be expected that we shall have the same rapid rate of decline in the future as in the past.

With regard to the reasons for the decline in phthisis mortality which has occurred, I listened with the utmost interest to Dr. Ransome's views.

I was greatly interested to see that he lays great stress upon the importance of land-drainage. Personally, I am unable to endorse that view. Some years ago I ventured to write on this subject*—after a careful examination of both Dr. Bowditch's and Sir George Buchanan's papers—and I came to the conclusion that the data did not completely support the inferences drawn from them; that, in other words, the data needed revising. I think, that if the same data were gone over again, brought up to date, and proper allowances made for social conditions, and for age- and sex-distribution, the same results would probably not be obtained.

One has to remember, for instance, in comparing impervious with pervious soils, that it has long been regarded as almost a commonplace that it is more healthy to live on a pervious than an impervious soil. The result of that is, that the pervious soils are lived upon by persons of relatively high social position, and the impervious soils are lived upon by the poorer classes: so that, when you compare the people living upon these two kinds of soil, you are to some extent comparing persons of high social position with persons of low social position, and that is a matter of direct importance in producing conditions of phthisis. I think that question of soil needs to be reinvestigated.

Then, furthermore—as Sir Shirley Murphy has already told us—whilst these main-sewerage works were going on, manufactures were increasing by leaps and bounds, and wages were increasing. It is quite true that the price of

* "The Influence of Soil on the Prevalence of Pulmonary Phthisis," *Practitioner*, February, 1901.

food did not go down at once, after the Corn Laws were repealed — indeed, not for ten or fifteen years. But, although food did not immediately become cheaper, the means of buying that food were increased, and consequently the social condition of the people was improved at a much earlier date than the reduction in the price of food. You have, therefore, two factors at work: first, the improvement in the status of life, accompanied later by reduction in the price of food; and, secondly, subsoil drainage: and it is a question to which of these two we are to ascribe the effect which in some districts was produced. Personally, I should be more inclined to ascribe it to improved conditions of life, etc., than to the reduction of the subsoil water; though the latter may have some effect by diminishing the tendency to bronchial conditions which predispose to phthisis.

Then, with regard to public-houses, I venture to think that, inasmuch as there is as much drinking going on now per head of the population as in former times, one cannot speak with any degree of confidence of improved habits with regard to temperance. It may be there is more "soaking," though less drunkenness; and that may possibly have as bad an effect in lowering vitality as bouts of drunkenness, and may do as much to prepare the way for attacks of the tubercle bacillus. Then one has to remember that it is not simply a matter of taking alcohol; it is a question of the drinkers making the public-house their club. I think Dr. Niven is right in ascribing much importance to personal infection in the public-house.

I was greatly interested to hear that the system of notification was inaugurated in Manchester in 1892. I was under the impression that Dr. Niven was the first to propose such a system in Oldham; but, it is quite clear, that the first attempt was made at an earlier date in Manchester. Dr. Niven did not succeed in persuading the Oldham people to adopt his system of notification, though he subsequently got it adopted in Manchester, about six months after we had it in operation in Brighton; so that I may venture to claim that Brighton was the first to have notification of phthisis in actual operation; and, as I have already said, we now have more cases annually notified per thousand of population than in the one town in which compulsory notification of phthisis is in force.

I will conclude by again expressing my thanks to Dr. Ransome for his most valuable and suggestive Paper.

DR. SEATON: At this late period of the evening, I hardly like to make any lengthy remarks; but I should wish to say that the point of greatest interest to me in Dr. Ransome's Paper is his consistency of teaching. I profited by the lectures—of which he was mainly the author—that were inaugurated by the late Manchester and Salford Association; and one could find there, almost word for word, the passages in this Paper which bear out his teaching more than a quarter of a century ago. Not that Dr. Ransome has not been progressive—quite the contrary. It is to him that we owe some of the very best observations as to the operation of the conditions of dwellings upon the action of bacilli—he has made all these celebrated scientific observations.

I myself was very much impressed with the consistency with which Dr. Ransome upholds the doctrine which he calls "the unconscious campaign against phthisis." On the other hand, I was rather surprised when I heard Dr. Newsholme speaking about the soil question. It seems to me that he overlooked the fact that, in places where the improvement took place, the population was the same before and after. The difference was in the subsoil, not in the population. It was the drainage operations which brought about the change in the mortality from phthisis. I think opinion must have changed very much on this subject and that the change is partly explained by the fact that, amongst the teachers of medicine, especially of late years, a sufficient importance has not been attached to the classical observations of forty years ago. The observations to which I refer, namely, those on the effect of drainage of the towns—the effect of sewerage works in not only drying but cleaning the subsoil of towns, are, I venture to say, firmly believed in by almost all in this room.

There are many points that I should like to dwell upon, but it is too late now to make any lengthy remarks; and I have only to add that I join with other speakers in expressing my high appreciation of the very valuable *resumé* that Dr. Ransome has given us.

DR. ALLAN: There are two points that I would like to mention, which have been referred to by some of the speakers: one is the question of notification. I observe lately, in the reports of many medical officers who have advocated the system of notification in the past, that they do not seem to find it a success. I hope they will not

be disheartened because it is not a success at first. I am sure that if the matter is taken up properly, and the cases are followed up afterwards, most beneficial results will accrue. I find that, in following up cases that have been notified, we come across many conditions which require ameliorating: one especially, which is not an infrequent condition to find in houses which have been occupied by phthisical people, and houses which appear to be "phthisical" houses, is a condition of dampness. In spite of the drainage which has been carried out, we still have a number of houses left in which there are no damp courses at all: the removal of the subsoil water must have made these houses more dry; but there are still certain houses in which a certain amount of dampness exists.

I think we must recognise, too, that phthisis is becoming almost entirely a disease of the very poor; and it must not be forgotten that phthisis may be the cause of the poverty, as well as poverty being partially the cause of the disease; and, perhaps, if we can remove the conditions of poverty, we shall find that we have, to a great extent, removed the predisposing cause of the disease. Undoubtedly, so long as rents are high, and people have to live in crowded districts, with a very small amount of room and very bad ventilation, we can hardly expect to get rid of phthisis.

An interesting case was brought to my notice the other day, which shows the value of ventilation. It was in connection with a large police-barracks, where for a number of years they were continually having cases of phthisis, and also cases of all kinds of disease. At last, after persistently reporting it to headquarters for a long time, an improved method of ventilating the whole place was instituted. The result is, as the police-surgeon was telling me the other day, he has now hardly anything to do. I think people are coming to recognise more and more the value of fresh air.

I have made out just lately a list showing what the effect of sanatorium treatment is on phthisical patients as compared with those who have not had it; and I find that the number of people who take precautions after sanatorium treatment is just about double what it is in the case of those who have not had it, but were under a medical practitioner at home. They do not seem to be impressed with the necessity of taking precautions: of opening their windows, of adhering to methods of cleanliness, taking care of their sputum, and things of that sort.

One of the things that prevents notification in London is,

that we cannot get the cases away anywhere. There is no chance of sanatorium treatment; and unless we can get something of that kind, the elimination of phthisis in London, will certainly be very much hindered for many years to come.

DR. BULSTRODE: I should like to make one or two comments with regard to the periodical rises in the curve, showing the steady decline of pulmonary tuberculosis in this country. In 1855, there was an outbreak of influenza which might possibly be thought of as accounting for the increase of tuberculosis at that time. In 1866 there was the cotton famine, which might be held to have aided the increase during that year. In 1890 the recent prevalence of influenza began, but I find that in 1891 there was a much greater number of deaths from influenza than in 1890. There are many suggestions, but it does not seem to me that they fully explain the elevations..

With respect to the rise in phthisis rates in Ireland, Dr. Ransome appears to attach a very large amount of importance to the sanitary conditions, but it seems very doubtful whether the sanitary conditions would account for the increase. I can understand that the sanitary conditions are no better, but I think it is improbable that they are worse. Personally, I think the increase is more likely to be due to the people of Ireland having degenerated in physique and resistance, owing to the constant strain of emigration of the most fit, thus leaving behind the less resistant to reproduce, perhaps, a people with a distinct hereditary proclivity to the disease. I think that, on natural history grounds, that is the more scientific explanation.

THE PRESIDENT: I think the discussion must now draw to a close, especially as we still have to hear Mr. Cooper.

I was going to make a few observations as to the question of phthisis and the prevention of phthisis as it arises in factories and workshops, as my contribution to the discussion, but I am going to spare you this. Dr. Ransome has already touched upon some of the points, and he is past-master in these things, and his advice has been of great service in the ventilation of factories in this country.

The lines representing the male and female phthisis death-rates in England, if traced backwards for a few years, approach each other, although the male rate still remains the higher, as now; but in Scotland they cross. Dr. Ransome has stated that in the eighteenth century, the records available showed a much higher phthisis-rate.

Can he say whether, at that time the two English lines had crossed; that is, whether the female phthisis death-rate then exceeded the male rate, as it does now in Ireland, and did recently in Scotland?

In conclusion, I want to tender to Dr. Ransome, on behalf of the Society, our cordial thanks for his admirable Paper.

DR. RANSOME: May I answer your question first, Mr. President? I do not think that male and female mortality were separated in the statistics that I saw at the Statistical Society. I do not think they were separated into males and females, so that I cannot tell whether the females were worse in those days. I should think it likely they were, because in those days the males were far more engaged in outdoor work than the females.

I must thank you very heartily for having so kindly received my Paper, and for the exceedingly careful discussion you have given to it. One or two speakers have already answered one another, so that I will not allude to Dr. Newsholme's remarks about drainage. I think he was answered by Dr. Seaton.

I did speak about the fact that drunkards' homes (including the starvation of their families), were a very potent cause of phthisis. Perhaps I have not laid sufficient stress upon the great harm of sheer poverty. I quite agree with everything that has fallen from the different speakers on that point. At the same time, mere poverty will not cause phthisis. I do not think the cause of it is, as the French say, "la misère physiologique," nor that it is due simply to deprivation of nourishment and deprivation of all sorts of other stimulants of life. It has been proved that the very poor people on the west of Scotland—on the Island of St. Kilda and the Western Hebrides—did not suffer from phthisis, were entirely free from it, and yet they were miserably housed and miserably fed. There certainly was sufficient ventilation there, whereas in the poverty-stricken homes in England there is no ventilation. Mere filth, again, would not do it, because in St. Kilda they simply lived upon a floor of ordure.

I do not lay the smallest stress upon the periodicity of the curves. I think it merely worth while noting that the period does come somewhat regularly about every twelve or thirteen years. There does seem to be some slight indication of this.

I must again thank you for your very kind reception of my Paper.

ANCIENT THEORIES OF CAUSATION OF FEVER BY MOSQUITOES.

The following Note by His Excellency the Governor of Ceylon has been forwarded to me by Sir Allan Perry, the Society's Colonial Secretary for that colony, and it is of so much historical interest that I am reproducing it in the *Transactions*. I have, unfortunately, been unable to secure the Sanskrit type, and have therefore had to be content with the reproduction of the translation.

H. TIMBRELL BULSTRODE.

BY HIS EXCELLENCY SIR HENRY A. BLAKE, G.C.M.G., ETC.,
Governor of Ceylon.

IN the course of inquiries into the causation of an epidemic of malarial fever in Mutwal, a division of the town of Colombo, last year, I received a report from fifteen members of the Sinhalese Medical Association, in which it was mentioned incidentally that, according to ancient authorities on Ayurvedic medicine, the chief causes of the disease are impure air and water and the existence of mosquitoes. I requested more precise information showing that mosquitoes were associated with the causation of malarial fever; and in reply I received extracts from ancient Indian works, among them an extract from Susruta—a Sanskrit work compiled, I am informed, from one of the lost Vedas. The age of the work is not known; but, as it was referred to in the works of a contemporary of Kálidása, the great Sanskrit poet who flourished in the sixth century of the Christian era, the work is at least 1,400 years old.

The following are the translations of the passages. They are taken from the Sanskrit edition published in 1873, and will be found at pages 43 and 45 (Kalpasthana) :—

KITA.

Life-destroying terrific twelve kind:

Tunginasa	Mandalapuchchhaka
Vichilaka	Tunganabha
Talaka	Sarshapika
Bahaka	Aralguli
Koshtagara	Sambuka
Kirimikara	Agnikita

Their bite is as painful as that of serpents, and causes diseases resulting from the three humours joined together; the bite, as if burnt with caustic or fire, is red, yellow, white, and pink colour, accompanied by fever, pain of limbs, hair standing on end, pain, vomiting, diarrhœa, thirst, heat, giddiness, yawning, shivering, hiccup, burning sensation, intense cold, vesicles or pustules increasing, swelling, knots under the skin, circles, etc.

Masakáh (Mosquitoes) are of five kinds :

Samudra	Krishna
Parimandala	Parwatiya
Hastimasaka	

Their bite causes swelling on the part bitten, itching. As for the parwatiya, it has similar qualities to life-taking insects.

H. SUMANGALA, High Priest,
Principal, Vidyódaya College, Maligakanda.

M. ÑANISSARA,
Assistant to the Principal, Vidyódaya College

I. SARVESWARASARMA, Brahmin.

SIMON DE SILVA, Gate Mudaliyar,
Chief Sinhalese Translator to Government.

R. C. KAILASAPILLAI, Mudaliyar,
Chief Tamil Translator to Government.

That the translation should be verified satisfactorily, I invited five Sanskrit scholars of acknowledged authority to meet at Queen's House. The translation is that unanimously adopted by these five gentlemen, who have signed the extract.

These passages, written possibly three thousand, and at least fourteen hundred years ago, are of singular interest, foreshadowing as they do the great discoveries of Manson and Ross.

Truly there is no new thing under the sun.

The Ordinary Meetings of the Society are held at

11, CHANDOS STREET, CAVENDISH SQUARE,

ON THE THIRD FRIDAY OF EACH MONTH FROM NOVEMBER TO MAY INCLUSIVE, UNLESS OTHERWISE ANNOUNCED.

THE CHAIR IS TAKEN AT HALF-PAST EIGHT O'CLOCK P.M. PRECISELY.

Subscriptions may be paid to the Treasurer. The Annual Subscription of Ordinary Members is One Guinea.

Ordinary Members are entitled to all privileges of membership, and to receive a copy of all the Society's publications issued during the year for which their subscriptions are paid. But Ordinary Members, resident beyond the limits of the Metropolitan Postal District, and having paid one or more annual subscriptions, may, on expressing to the Hon. Treasurer a wish to that effect, be distinguished as Non-Resident Members.

Non-Resident Members are liable to an annual payment of half-a-guinea. They have the privilege of attending meetings, and are entitled to receive the publications of the Society issued during the year for which their subscription is paid; but they are not entitled to vote at meetings of the Society nor to hold office in it.

The Rules and Regulations, etc., of the Society, with an Exposition of its Objects, may be obtained from the Secretaries.

The Transactions for the Sessions 1859-60, 1866-67, *and* 1867-68 *are out of print. With these exceptions, those for the Sessions from* 1860-61 *to the last Session inclusive, may be obtained from the publishers,* Messrs. WILLIAMS AND NORGATE, 14, HENRIETTA STREET, COVENT GARDEN, LONDON, W.C. *Prior to* 1859-60, *the Transactions were published in the* "SANITARY REVIEW." *The issue of a New Series of the Transactions was begun in* 1882.